Through the looking glass

Smink, Frédérique Rebecca Esther

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Epidemiological studies on eating disorders in primary care and the community

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Chapter 1

Introduction

This thesis aims to explore several aspects of the epidemiology of eating disorders in primary care and the community, with an emphasis on the effects of changed diagnostic criteria, a changing sociocultural environment over time, and the impact of self-perceived and peer-perceived social status on the occurrence of eating disorders. The following paragraphs provide a background to the topics and research questions addressed in this thesis.

EATING DISORDERS

Eating disorders are severe mental health problems with detrimental consequences for physical and psychosocial health.[1,2] In a meta-analysis of excess mortality in the 1990s,[3] the eating disorder anorexia nervosa (AN) was associated with the highest rate of mortality among all mental disorders; according to a recent meta-analysis, one out of twenty AN patients will have died after a decade.[4]

The core pathology of the most well-known eating disorders AN and bulimia nervosa (BN) is an overvaluation of weight and shape.[1] Most people derive their sense of self-worth and self-esteem from different sources, such as their competence at work or at school, interpersonal relationships, athletic achievements, etcetera. In eating disorder patients, however, self-esteem is entirely dependent on their weight and body shape.

Another central feature of both AN and BN is dietary restriction.[1] In individuals with AN, this results in underweight, or – in children and adolescents – a failure to meet expected increases in weight and growth according to age.[2] People with BN, on the other hand, who are by definition of normal weight or overweight, at length fail in their attempt to restrict their food intake, resulting in binge eating followed by compensatory behaviors to prevent weight gain. The essence of a binge-eating episode is the subjective sense of loss of control over eating;[5] the sense that one cannot stop eating until all available food is consumed, or one has to stop because of severe abdominal discomfort. During a binge-eating episode, often large amounts of palatable foods, rich in sugar and/or fat, are rapidly consumed. Though binge eating can initially provide relief from negative feelings and food craving,[6] an episode of binge eating is invariably followed by feelings of regret, guilt and shame. Strategies to counteract the anticipated consequences of binge eating – primarily weight gain - encompass purging, including self-induced vomiting and the use of laxatives and diuretics, and non-purging compensatory behaviors, such as excessive and compulsive exercise, or prolonged fasting. Since fasting increases the risk

for binge eating, a vicious cycle is thus created. Binge eating and purging can also occur as a symptom of AN, referred to as AN of the binge-purge subtype. In the other AN subtype, only restriction of intake is prominent. AN patients often have a markedly disturbed body image: they experience their body as fat, even when body weight is dangerously low.[2]

Eating disorders are usually classified according to the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM). A reliable guideline to classify mental disorders offers a universal and transparent language to clinicians to inform treatment decisions in an individual patient; besides, it is an essential tool to examine the prevalence of disorders to guide health care policy, to select patient groups for scientific study purposes, and to document morbidity and mortality rates.[2] The fifth edition of the DSM, DSM-5, was released in May 2013,[2] almost two decades after publication of the previous edition, the DSM-IV, in 1994.[7] In those two decades, a wealth of research in the fields of cognitive neuroscience, neuroimaging, genetics and epidemiology has expanded our knowledge of mental disorders. Thus, an important limitation of the DSM-IV was uncovered: it is too rigid a categorical system. Clinical and scientific observations have shown that the boundaries between certain disorder categories are not clear cut at all, and that some symptoms are not bound to one disorder, but can occur with varying severity in a whole range of disorders.[2] This limitation of the DSM-IV also pertains to the section on eating disorders (e.g., Fairburn & Cooper[8] and Attia et al.[9]).

The DSM-IV specifies only two eating disorders, AN and BN.[7] In DSM-5, the significantly revised eating disorder section specifies three eating disorders: AN, BN, and binge-eating disorder (BED).[2] In BED, binge-eating episodes are – in contrast to BN – not followed by compensatory behavior. Therefore, BED is often seen in obese individuals. The decision to establish BED as a mental disorder was not without controversy. A major complaint – not limited to BED – about classifying certain behaviors as mental disorders is that behaviors that may seem quite common (such as binge eating) are unnecessarily medicalized.[10] Compared to obesity per se, however, binge eating is associated with higher levels of psychopathology and weight- and shape concerns, and lower quality of life.[11] Obesity on the other hand, though also considered for the status of a mental disorder,[12,13] was eventually not included in the DSM-5 (as it had not been in the DSM-IV either) because evidence that obesity is primarily caused by a mental dysfunction was unconvincing.[13] It is nevertheless intriguing to imagine the immense – and probably unaffordable – mental health care policy implications of such a status, considering the fact that 35% of the adult US population is obese,[14] and would thus be diagnosed

Table 1.1 Severity rating of eating disorders in DSM-5

	AN	BN	BED
Mild	BMI ≥ 17	1–3 episodes of inappropriate compensatory behaviors per week	1–3 BE episodes per week
Moderate	BMI 16–16.99	4–7 episodes per week	4–7 BE episodes per week
Severe	BMI 15–15.99	8–13 episodes per week	8–13 BE episodes per week
Extreme	BMI <15	14 or more episodes per week	14 or more BE episodes per week

AN: anorexia nervosa; BN: bulimia nervosa; BED: binge-eating disorder;
BMI: body mass index (kg/m^2); BE: binge eating

with a 'new' mental disorder. Hence, it is an important question how changes in diagnostic criteria and categories affect the number of people with a mental disorder diagnosis; in this case, with an eating disorder diagnosis.

A new feature in the DSM-5 is the introduction of a severity rating for disorders, ranging from mild to extreme, in order to help clinicians delineate treatment and track a patient's progress. Key severity parameters for eating disorders have been defined (see Table 1.1). Clinicians can increase severity ratings based on the presence and severity of other symptoms, and the degree of functional disability.[2] The validity of the severity ratings for eating disorders is unknown.

EPIDEMIOLOGY

Epidemiological studies provide information about the occurrence of disorders. Morbidity and mortality rates derived from epidemiological studies inform health care policy and the planning of treatment services. Incidence studies may shed light on risk factors. As mentioned before, a reliable classification is a *sine qua non* for this purpose. The other way round, epidemiological data can also shape the development of a classification. The decision to establish BED as a specific eating disorder, for example, was partly informed by epidemiological data, proving its construct validity by showing a distinct profile of clinical characteristics and correlates of BED compared with the other eating disorders.[11]

Longitudinal epidemiological studies investigate trends in the frequency of disorders over time. To reliably examine time trends in morbidity rates, it is essential that the classification used does not change over time. Differences in definitions of disorder make comparisons between studies difficult – if not impossible –, and prove to be a major challenge for long-standing (e.g., spanning decades) epidemiological studies.[15] Time trends in morbidity rates provide clues to the etiology

of a disorder, especially to sociocultural factors, as it is assumed that the biological make-up of a species does not change within a few decades. Thus, epidemiological studies examining secular trends may uncover risk factors that affect the society at large. In the case of eating disorders, this knowledge is salient since sociocultural factors are thought to play a major role.[16]

The etiology of eating disorders is relatively poorly understood.[17,18] Although many risk factors have been described,[19] the exact mechanism through which an individual develops an eating disorder is unknown.[1] Moreover, though present in virtually all eating disorder patients, the risk factor body dissatisfaction – widespread among women in the Western world – is hardly sufficient to develop an eating disorder.[20]

In accordance with most other mental disorders,[2] risk factors for eating disorders are categorized along social, psychological and biological lines (e.g., Jacobi. et al.[19]); in other words: from influences from the society at large and the social environment to the psychological and biological characteristics of the individual itself. Examples of societal risk factors include living in a Western culture, which places great value on the thin-body ideal, especially for women.[16] This thin-body ideal is promoted and reinforced through the media[16] and through 'micro societies' such as family and peers.[21,22] Many girls and women feel that their bodies do not adhere to this standard and develop body dissatisfaction.[23] Psychological risk factors include low self-esteem, perfectionism (especially in AN), anxiety and depression, and difficulties with identity and autonomy development; salient processes in adolescence.[16,19,20] Biological factors include a genetic predisposition (e.g., female sex) and neuroendocrine disturbances, for example in the central control of appetite.[17]

A factor associated with a certain disorder may only be called a risk factor if it is established that the factor in question precedes the outcome. Prospective, longitudinal studies are the gold standard to examine precedence.[19] This type of study is relatively scarce in the field of eating disorders, because longitudinal studies, especially for such low-prevalent disorders, are expensive and time-consuming to conduct. Moreover, to distinguish consequences or concomitants of a disorder from true risk factors, a longitudinal study would ideally have to start before onset of the disorder. Since eating disorders usually develop in adolescence,[15] early adolescence would be an advantageous starting point for longitudinal research into putative risk factors. While examining time trends may provide knowledge about risk factors on a macro (societal) level, studying a cohort of early adolescents may offer insight into sociocultural risk factors on a micro level (e.g., at school or at home). The micro environment of – future – eating disorder patients has been

studied before (e.g., Polivy & Herman[20]), but mostly through self-report, which may be biased.[24] Therefore, a call for multiple informant data (e.g., peers) in eating disorder research has been made.[24]

OUTLINE AND SCOPE OF THIS THESIS

Research questions addressed in this thesis include: Has the incidence of eating disorders changed over time? How do changes in the diagnostic criteria from DSM-IV to DSM-5 affect the number of people with an eating disorder diagnosis? Furthermore, an attempt is made to advance our understanding of how eating pathology develops by examining the role of self-perceived and peer-perceived social status in early adolescence as a potential risk factor.

Chapter 2 provides a comprehensive review of the literature on the epidemiology of eating disorders, focusing on the basic epidemiological parameters incidence, prevalence and mortality rate.

Chapter 3 examines changes in the incidence of anorexia nervosa and bulimia nervosa in the Netherlands during the 1980s, 1990s and 2000s, using data from a nationwide network of general practitioners, serving a representative sample of the total Dutch population.

Chapter 4 discusses the literature on epidemiology, course, and outcome of eating disorders in accordance with the DSM-5. The first part describes the consequences of the revised diagnostic criteria for the incidence and prevalence of anorexia nervosa, bulimia nervosa, binge-eating disorder, and the residual diagnosis of not otherwise specified eating disorders. The second part reviews course and outcome studies regarding the three specific eating disorders in DSM-5 – anorexia nervosa, bulimia nervosa and binge-eating disorder.

Chapter 5 reports on the prevalence and severity of DSM-5 eating disorders in a community cohort of Dutch adolescents. The validity of severity ratings is examined by linking severity level to detection and treatment rates by (mental) health care.

Chapter 6 investigates whether self-perceived and peer-perceived social status in early adolescence is associated with eating pathology in young adulthood. Data from the same community cohort of Dutch adolescents as described in Chapter 5 are used.

Chapter 7 provides a general discussion of the main findings of this thesis.

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Chapter 2

Epidemiology of eating disorders: incidence, prevalence and mortality rates

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ABSTRACT

Eating disorders are relatively rare among the general population. This review discusses the literature on the incidence, prevalence and mortality rates of eating disorders. We searched online Medline/Pubmed, Embase and PsycINFO databases for articles published in English using several keyterms relating to eating disorders and epidemiology. Anorexia nervosa is relatively common among young women. While the overall incidence rate remained stable over the past decades, there has been an increase in the high-risk group of 15-19-year-old girls. It is unclear whether this reflects earlier detection of anorexia nervosa cases or an earlier age at onset. The occurrence of bulimia nervosa might have decreased since the early nineties of the last century. All eating disorders have an elevated mortality risk; anorexia nervosa the most striking. Compared with the other eating disorders, binge-eating disorder is more common among males and older individuals.

INTRODUCTION

Epidemiological studies provide information about the occurrence of disorders and trends in the frequency of disorders over time. For epidemiological studies on eating disorders there are some methodological issues. Eating disorders are relatively rare among the general population and patients tend to deny or conceal their illness and avoid professional help. This makes community studies costly and ineffective. Therefore, many epidemiological studies use psychiatric case registers or medical records from hospitals in a circumscribed area. This type of study will underestimate the occurrence of eating disorders in the general population, because not all patients will be detected by their general practitioner or referred to the hospital or mental health care. Furthermore, differences in rates over time could be due to improved case detection, increased public awareness leading to earlier detection and wider availability of treatment services, instead of a true increase in occurrence.[1,2]

Anorexia nervosa (AN) and bulimia nervosa (BN) are the two specified eating disorders according to the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV). However, the most common eating disorder diagnosis in either clinical and community samples is the rest category 'eating disorder not otherwise specified' (EDNOS).[3-7] EDNOS is a heterogeneous, not well defined group of eating disorders and includes partial syndromes of AN and BN, purging disorder and binge-eating disorder (BED). A comprehensive meta-analysis of 125 studies suggests that EDNOS is associated with substantial psychological and physiological morbidity, comparable with the specified eating disorders.[8] In 2013 the fifth edition of the DSM is scheduled to appear, including a thoroughly revised eating disorder section. A major goal is to reduce the size of the EDNOS-category. To achieve this goal the criteria for AN and BN will be broadened[9,10] and BED will be added as a specific eating disorder. The decision to make BED a separate diagnosis is partly informed by epidemiological data supporting the construct validity of BED. BED differs from AN and BN in terms of age at onset, gender and racial distribution, psychiatric comorbidity and association with obesity. BED is often seen in obese individuals, but is distinct from obesity per se regarding levels of psychopathology, weight- and shape concerns and quality of life.[11] BED aggregates strongly in families independently of obesity, which may reflect genetic influences.[12,13]

In this review we will describe the epidemiology of AN, BN, EDNOS and BED according to DSM-IV and – if available- to the proposed DSM-5 criteria. The pro-

posed changes in DSM-5 diagnostic criteria will alter the coverage of the diagnostic categories and thus their disease frequencies as well. Some studies used both a narrow and a broad or partial definition of AN, including DSM-IV AN with or without amenorrhea and ICD-10 atypical AN.[14-16] These broad or partial definitions of AN are in line with the proposed DSM-5 criteria for AN and will be referred to as 'broad AN' throughout this review.[9] In a Finnish study of female twins, the 5-year clinical recovery rates of AN and broad AN were almost the same; i.e. 66.8% and 69.1% respectively, providing evidence for the validity of broad AN.[14] Definitions of each epidemiological measure are provided at the respective paragraphs.

This article is based on research publications on the epidemiology of eating disorders and updates our previous reviews, with special emphasis on studies published in the last three years.[2,17-19]

METHOD

We searched online Medline/Pubmed, Embase and PsycINFO databases using several keyterms relating to eating disorders and epidemiology. The reference lists of the articles found were checked for any additional articles missed by the database search. This review is limited to articles published in English, describing the basic epidemiological parameters incidence, prevalence and mortality rates.

Incidence

The incidence rate is the number of new cases of a disorder in the population over a specified period. The incidence rate of eating disorders is commonly expressed in terms of per 100,000 persons per year (person-years). The study of new cases provides clues to etiology.

Anorexia nervosa

Community studies assessing the incidence of eating disorders are scarce. Keski-Rahkonen and colleagues conducted a large community study to quantify the incidence of AN, yielding an incidence rate of 270 per 100,000 person-years in 15-19-year-old Finnish female twins during 1990-1998.[14,19] The incidence rate of broad AN was 490 per 100,000 person-years in the same group.[14] A much higher incidence rate of 1,204 per 100,000 person-years (95% confidence interval (CI): 652-2,181) for broad AN in females aged 15-18 was found in another Finnish study of a relatively small sample of 595 adolescents.[20] The high incidence rate might be

explained by the small sample size limiting statistical power and a very broad definition of AN used in this study, including subjects with an age-adjusted body mass index (BMI) up to 19, without explicitly stating that weight loss of at least 15% had to be present. Community rates are much higher than incidence rates derived from primary care and medical records,[1,21] reflecting the selection filters that form the pathway to (psychiatric) care.[22]

Incidence rates derived from general practices represent eating disorders at the earliest stage of detection by the health care system. Currin and colleagues[23] searched the General Practice Research Database in the UK for new cases of AN between 1994 and 2000 and compared their data with the findings of a similar study for 1988-1993.[24] The age-adjusted and sex-adjusted incidence rate of AN remained stable over the two study periods: in 2000 it was 4.7 (95% CI: 3.6-5.8) per 100,000 person-years, compared with 4.2 (95% CI: 3.4-5.0) per 100,000 person-years in 1993. In the Netherlands the overall incidence rate of AN ascertained by general practitioners in a large representative sample of the Dutch population remained stable as well. In 1995-1999 it was 7.7 (95% CI: 5.9-10.0) per 100,000 person-years, practically the same as the rate of 7.4 per 100,000 person-years in 1985-1989.[1] Incidence rates are highest for females aged 15-19 years. They constitute approximately 40% of all cases, resulting in an incidence rate of 109.2 per 100,000 15-19-year-old girls per year in 1995-1999.[1] The incidence of AN among males was less than 1 per 100,000 person-years in general practices in the Netherlands and the UK.[1,23] AN does occur among children <13 years of age, but is relatively rare.[1,23] Three studies used a national Paediatric Surveillance System to identify new cases of early-onset eating disorders presenting to pediatricians.[25-27] In Canada, the incidence rate of early-onset restrictive eating disorders diagnosed by pediatricians was 2.6 (95% CI: 2.1-3.2) per 100,000 person-years in children aged 5 to 12 years;[25] in Australia it was 1.4 (95% CI: 1.1-1.7) per 100,000 person-years in 5-13-year-old children.[27] In the Canadian study 62% of new restrictive eating disorder cases met criteria for AN.[25] Of the Australian pediatric inpatients with a newly diagnosed restrictive eating disorder only 37% could be classified as AN, although 61% had life- threatening complications of malnutrition.[27] In British pediatric and psychiatric care an overall incidence rate of 1.1 per 100,000 person-years for AN was found among children <13 years of age.[26] Among middle aged and elderly women AN is relatively rare as well.[28-30] In a Spanish population-based study using the Public Health Registry to identify eating disorder cases diagnosed by mental health professionals, new cases of AN were found among women over 45 years of age, constituting 64% of all new

eating disorder diagnoses in this age-group.[31] It is unknown whether this reflects late detection or late age at onset.

The question of whether the incidence of AN is on the rise has been under debate. Long-term epidemiological studies are sensitive to minor changes in the absolute incidence numbers and in the methods used, for example, variations in registration policy, demographic differences between the populations, faulty inclusion of readmissions, the specific methods of detection used or the availability of services.[18,32] In a meta-analysis of the incidence of AN in mental health care, various studies in northern Europe were combined (see Figure 2.1). Until the 1970s, there was an increase of the registered incidence of AN in Europe. Since 1970, the incidence of AN in Europe seems to have been rather stable.[1,18,33,34] In Switzerland the incidence of severe AN in females was studied in a geographically defined region using the same methodology from 1956 to 1995. The incidence of severe AN requiring hospital admission rose significantly during the 1960s and 1970s and reached a plateau of around 1.2 per 100,000 person-years thereafter.[21]

In the Netherlands from the 1980s up to now general practitioners have registered new cases with an eating disorder in a representative sample of the Dutch population. While the overall incidence of AN was stable around 7 per 100,000 person-years, the incidence in 15-19-year-old girls increased significantly from 56.4 per 100,000 person-years in 1985-1989 to 109.2 per 100,000 person-years in 1995-1999.[1] This is in line with an Italian study examining age at onset of AN in a large sample of 1,666 patients referred to an eating disorders outpatient unit between

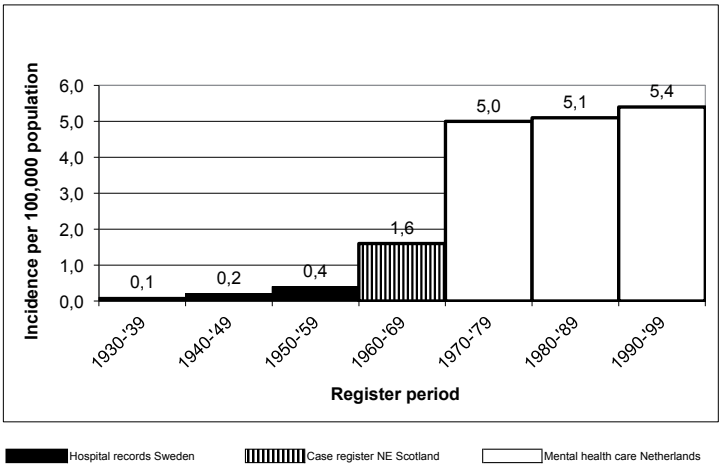

Figure 2.1 Registered yearly incidence of anorexia nervosa (adapted from Hoek[18])

1985 and 2008. Patients referred in more recent years had an earlier age at onset.[35] In Rochester, MN, USA, the age-adjusted incidence rates of AN showed a significant linear increasing trend only in females aged 15-24 years from 1935-1989.[36]

Bulimia nervosa

Only a few incidence studies of BN have been conducted. In the community study of the 1975-1979 birth cohorts of female Finnish twins the incidence rate of BN was 200 per 100,000 person-years at the peak age of incidence, 16-20 years.[37] A broader definition of BN was examined as well. When symptom frequency was relaxed to once a week, in concordance with the proposed DSM-5 criteria,[10] the peak incidence rate of broad BN was 300 per 100,000 person-years in 16-20-year-old females.[37] Isomaa and colleagues found an incidence rate of 438 (95% CI: 132-1,175) per 100,000 person-years in 15-18-year-old Finnish females for another broad definition of BN, including subjects who fulfilled all but one of the criteria for BN.[20]

According to the nation-wide primary-care study in the Netherlands, the overall incidence rate of BN tended to decrease from 8.6 per 100,000 person-years in 1985-1989 to 6.1 per 100,000 person-years in 1995-1999.[1] In a primary-care study from the UK, the overall age- and sex-adjusted incidence rate of BN decreased during the second half of the 1990s from 12.2 per 100,000 person-years in 1993 to 6.6 per 100,000 person-years in 2000. However, the incidence rate of BN in women aged 10-19 years remained relatively stable around 40 per 100,000 person-years in 1993 as well as in 2000.[23]

Several studies suggest that age at onset of BN is decreasing. In a sample of 793 Italian BN patients referred to an eating disorders outpatient unit between 1985 and 2008, subjects born in 1970-1972 had a mean age at onset of 18.5 years, compared to 17.1 years in subjects born between 1979-1981.[35] In the Dutch primary-care study the high-risk group of BN shifted from 25-29-year-old females in 1985-1989 to 15-24-year-old females in 1995-1999.[1] It is unclear whether this reflects a true earlier age at onset or rather earlier detection of BN cases.

Eating disorder not otherwise specified and binge-eating disorder

Epidemiological studies on EDNOS are sparse, because of its heterogeneity and undefined operational criteria, except for BED, for which in DSM-IV research criteria were formulated. In a Spanish population-based study using the Public Health Registry to identify eating disorder cases diagnosed by mental health professionals, the incidence rate of EDNOS was 6.5 (95% CI: 4.8-7.9) per 100,000 inhabitants per year.[31] A British national surveillance study of newly diagnosed

eating disorders in pediatric and psychiatric care found an incidence rate of 1.2 per 100,000 person-years for EDNOS among children <13 years.[26] To our knowledge no incidence studies on BED yet exist. Binge eating as a disordered eating behavior or symptom is quite common among adolescents: in a longitudinal study of a large cohort of US adolescents, the incidence rate for binge eating was 10.1 per 1,000 person-years years among females and 6.6 per 1,000 person-years among males (both sexes ≥14 years), which translates into 1,010 per 100,000 person-years and 660 per 100,000 person-years among female and male adolescents, respectively.[38]

Prevalence

The prevalence can be expressed as point prevalence, one-year prevalence rate and lifetime prevalence. The point prevalence is the prevalence at a specific point in time, e.g., January 1 of a specific year. The one-year prevalence rate is the point prevalence plus annual incidence rate (the number of new cases in the following year). The lifetime prevalence is the proportion of people that had the disorder at any point in their life. The prevalence is the most useful measure for planning health care facilities, as it indicates the demand for care. Case detection through a two-stage screening approach is the standard procedure to estimate the prevalence of eating disorders.[2,39] In the first stage a large population is screened for the likelihood of an eating disorder by means of a screening questionnaire that identifies an at-risk group. In the second stage definite cases in the at-risk group are established on the basis of a personal interview. Problems associated with this type of study are poor response rates, sensitivity of the screening instrument and the restricted size of the groups interviewed.[40] To circumvent this last problem several studies use a structured interview such as the Composite International Diagnostic Interview (CIDI), usually administered by lay-interviewers, to assess the prevalence of eating disorders in a large population sample.

Anorexia nervosa

The lifetime prevalence of AN and broad AN has been assessed in three large population-based cohort studies of twins.[14-16] In Sweden, it was 1.2% (AN) and 2.4% (broad AN) in the largest twin study of women from the 1935-1958 birth cohorts.[16] In an Australian study of female twins aged 28-39 years, the lifetime prevalence of AN was 1.9% and of broad AN 4.3%.[15] The lifetime prevalence of AN was 2.2% and of broad AN 4.2% in a large sample of women from the 1975-1979 birth cohorts of Finnish twins.[14] In men from the same birth cohort the lifetime prevalence of AN was 0.24%.[41]

Stice and colleagues followed a relatively small sample of 496 adolescent girls over an 8-year period from early adolescence into young adulthood, administering annual diagnostic interviews. They found a lifetime prevalence by age 20 years of 0.6% for AN and 2.0% for broad AN.[42] In Portugal, the point prevalence of AN among adolescent girls was 0.39% and of broad AN 0.64%.[6] In an Australian population-based sample of 1,597 14-year-old boys and girls, only one case of AN was identified by means of a self-report eating disorder screening questionnaire; four other subjects met partial criteria for AN.[43]

Prevalences of AN estimated with two-stage procedures varied from 0 to 0.9% with an average point prevalence of 0.29% in young females.[2] In a meta-analysis,[2] the one-year prevalence rate per 100,000 young females was computed at different levels of care (Table 2.1). Using two-stage studies of community samples and estimates of the incidence, the one-year prevalence rate of AN in the community was calculated as 370 per 100,000 young females. One can conclude from Table 2.1 that the majority of patients with AN in the community do not enter the mental health care system.[18]

Several studies used the CIDI to estimate the lifetime prevalence of AN in large population samples.[45-47] Both in a nationally representative survey of the US household population[45] and in a population-based study in six European countries,[46] the lifetime prevalence of AN was 0.9% among adult females. In the US study it was 0.3% among males,[45] while in the European study not a single male case of AN was found.[46] In a large representative sample of US adolescents the lifetime prevalence of AN was 0.3% in 13-18-year-old females as well as males.[47] The female-to-male ratio in these studies is considerably lower than the 10:1 ratio found in the Finnish twin study and as reported in previous reviews,[2,14,41] which could be due to differences in methodology and small numbers of cases with eating disorders.[45,47] However, despite this restriction many recent community-based studies have found that AN is more common among males than previously thought. AN may be even more frequently underdetected in males than in females.[19]

Table 2.1 Estimates of one-year prevalence rates per 100,000 young females at different levels of care

Level of morbidity	Anorexia nervosa	Bulimia nervosa*
Community	370	1000
Primary care	160	150
Mental health care	127	75

* Based on a meta-analysis of Hoek and van Hoeken[2] in 2003 and corrected for more recent findings of a decrease in point prevalence[44] and incidence[1] of BN

A large study of Swedish twins born during the period 1935-1958 documented a higher prevalence of AN in both male and female participants born after 1945 than those born before 1945.[16]

Bulimia nervosa

The generally accepted point prevalence of BN from two-stage studies is about 1% among young females.[2,40] Keski-Rahkonen and colleagues found a lifetime prevalence of 1.7% for BN in women from the 1975-1979 birth cohorts of Finnish twins.[37] When symptom frequency was relaxed to once a week, in concordance with the proposed DSM-5 criteria, the lifetime prevalence rose to 2.3% in women.[37] In an Australian twin cohort of women aged 28-39 years, a lifetime prevalence of BN of 2.9% was found.[15] According to US[45] and European[46] large-scale two-stage studies of the population the lifetime prevalence of BN, assessed with the CIDI, varied between 0.9 and 1.5% among women and between 0.1% and 0.5% among men. Marques and colleagues compared the prevalence of BN across nationally representative samples of ethnic groups in the US. BN was more prevalent among Latinos and African Americans than non-Latino whites. Lifetime prevalences ranged from 0.51% (non-Latino whites) to 2.0% (Latinos).[48] In a recent study of a nationally representative sample of US adolescents, a lifetime prevalence of BN of 1.3% and 0.5% was found among 13-18-year-old females and males, respectively.[47] In a US sample of 496 adolescent females, followed for 8 years, a lifetime prevalence of 1.6% for BN was found at age 20 years.[42] An Australian population-based study of 1,597 14-year-old adolescents reported 9 cases of BN, translating into a point prevalence of 0.6%.[43]

Trace and colleagues assessed the impact of reducing the binge-eating frequency on the lifetime prevalence of BN in a large population sample of female Swedish twins. The lifetime prevalence of BN increased from 1.2% for a minimum of 8 binges per month (DSM-IV) to 1.6% for at least 4 binges per month (proposal DSM-5).[49]

The decrease in occurrence of BN over time found in the incidence studies is supported by a US study of university students in which the point prevalence of BN among women significantly decreased from 4.2% in 1982, to 1.3% in 1992 and 1.7% in 2002.[44] In another US study among female students the point prevalence of probable cases of BN remained relatively stable between 1990 and 2004.[50]

Eating disorder not otherwise specified and binge-eating disorder

Often used diagnostic interviews to estimate the prevalence of eating disorders, like the CIDI and the Structured Clinical Interview for DSM disorders (SCID) do not cover EDNOS. In recent studies that used the CIDI alterations have been made to include subthreshold AN[47] and BED.[45-47] Researchers have operationalized EDNOS in different ways; reported prevalences are therefore difficult to compare and in community studies the use of limited defintions will underestimate the true prevalence of eating pathology that could be classified as EDNOS.[8] The point prevalence of EDNOS in a nation-wide community sample of young females was 2.4%.[6]

The lifetime prevalence of BED has been assessed in large population samples in the US[45,47] and Europe.[46] In six European countries it was 1.9% for women and 0.3% for men.[46] In the US higher lifetime prevalences were found among adults (women 3.5%; men 2.0%) and among 13-18-year-old adolescents (girls 2.3%; boys 0.8%).[45,47] The US researchers used a duration criterium of only three months instead of the six months DSM-IV research criteria require, which might partly explain the higher percentages. Hudson and colleagues examined data from a non-clinical sample to estimate how much the prevalence of BED will increase under the proposed DSM-5 criteria that relax the requirements for the frequency (from two per week to one per week) and duration of binges (from six to three months). They extrapolated their findings to the results of the aforementioned study of the US household population and estimated that the lifetime prevalence of BED would increase with an additional 0.1% to 3.6% in women and 2.1% in men.[51] In a study of a large sample of adult Swedish female twins, a relatively low lifetime prevalence of 0.17% for BED was found, which rose to 0.35% when DSM-5 criteria were applied.[49]

Mortality

One could describe the mortality rate as an incidence rate in which the event being measured is death.[52] Mortality rates are often used as one of the indicators of illness severity. The standard measures for mortality are the crude mortality rate (CMR) and the standardized mortality ratio (SMR). The CMR is the number of deaths within the study population over a specified period. The SMR is the ratio of observed deaths in the study population to expected deaths in the population of origin.[18,19,52]

Anorexia nervosa

In a meta-analysis of excess mortality in the 1990s, AN was associated with the highest rate of mortality among all mental disorders.[53] In a recent meta-analysis of 35 published studies,[54] the weighted CMR for AN was 5.1 deaths (95% CI: 3.99-6.14) per 1,000 person-years, translating into 5.1% per decade or 0.51% per year. One in five individuals with AN who died had committed suicide.[54] The overall SMR was 5.86 (95% CI: 4.17-8.26) with a mean follow-up period of 14 years. The duration of follow-up is inversely correlated with the reported SMR; as duration of follow-up increases, the expected mortality in the population of origin will increase as well, resulting in lower SMRs. In a meta-analysis of SMRs in 2001, the overall SMR of AN in studies with 6-12 years of follow-up was 9.6 (95% CI: 7.8-11.5) and in studies with 20-40 years of follow-up 3.7 (95% CI: 2.8-4.7).[55] Age, case severity and study period influence mortality rates as well.[54] In a Swedish study,[56] a significantly higher mortality rate (4.4% vs. 1.2%) was found among female patients hospitalized due to AN in 1977-1981 compared with those hospitalized in 1987-1991. The authors argue that this dramatic decrease in mortality is related to the introduction of specialized care units for patients with eating disorders. Finally, in an audit conducted in the UK, death certificates emerged as a flawed source of information with both over- and underreporting of AN as a cause of death, the latter probably more common.[19,57]

Bulimia nervosa

In a recent meta-analysis of 12 studies describing the mortality rates of patients with BN, a weighted mortality rate of 1.74 per 1,000 person-years was found (95% CI: 1.09-2.44), which means that per year 0.17% of BN patients die. The overall SMR was 1.93 (95% CI: 1.44-2.59).[54] Crow and colleagues examined mortality in a sample of 1,885 patients evaluated for treatment for eating disorders at an outpatient clinic between 1979 and 1997. Of the 906 BN patients, 35 (3.9%) had died after a mean follow-up of almost 19 years. Suicide accounted for 23% of deaths.[58]

Eating disorder not otherwise specified and binge-eating disorder

A recent meta-analysis of six studies describing the mortality rate of patients with EDNOS reported a weighted annual mortality rate of 3.31 deaths (95% CI: 1.48-5.75) per 1,000 person-years. The overall SMR was 1.92 (95% CI: 1.46-2.52).[54] Any elevated mortality risk of EDNOS could be partly explained by the assertion that EDNOS sometimes reflects the earlier stages of AN.[54,59] None of the six studies stated that BED was part of the EDNOS category, which may be explained by the

fact that the study periods of these studies were (in part) before the publication of DSM-IV in 1994, in which BED was introduced.[58-63]

Keel and Brown reviewed six studies describing course and outcome of BED.[64] Duration of follow-up ranged from one (four studies) to 12 years (one study). The single 12-year follow-up study provided the only report of deaths at follow-up: 2 of 68 patients admitted for inpatient treatment of BED had died after 12 years, leading to a CMR of 2.9% and a non-significant SMR of 2.29 (95% CI: 0.00-5.45).[65] These data from an inpatient sample may not be representative of patients with BED seen in other settings.[64] BED is associated with obesity. In a large US population-based study 42% of the subjects with a lifetime diagnosis of BED were obese (BMI >30 kg/m^2) at the time of the interview and also a significantly higher prevalence of morbid obesity (BMI >40 kg/m^2) was found among these subjects compared to respondents without any eating disorder (OR 4.9; 95% CI: 2.2-11.0).[45] Obesity and especially morbid obesity is associated with increased risk for mortality, although the net effect of obesity on mortality is difficult to quantify.[66,67] Finally, in a meta-analysis of the risk of suicide in eating disorders, no suicide had occurred among 246 patients with BED after a mean follow-up of 5.3 years.[68]

Eating disorders in non-Western countries and among ethnic minorities

In the past, eating disorders have been characterized as culture-bound syndromes, specific to Caucasian subjects in Western, industrialized societies.[69] Recent studies demonstrate that eating disorders and abnormal eating behaviors do occur in non-Western countries and among ethnic minorities.[48,70-74] An increasing occurrence of eating disorders in non-Western countries has been associated with cultural transition and globalization, including modernization, urbanization and media exposure promoting the Western beauty ideal.[70,75-77] The most comprehensive attempt to quantify eating disorders in a non-Western setting was conducted on the Caribbean island Curaçao (Netherlands Antilles), where the full spectrum of community health and service providers was contacted. The overall incidence of AN of 1.82 (95% CI: 0.74-2.89) per 100,000 person-years was much lower than in the US and Western Europe. No cases were found among the black population. However, the incidence of 9.08 (95% CI: 3.71–14.45) among the minority mixed and white population was similar to the incidence in the Netherlands and in the United States.[78] In the Netherlands, incidence rates of psychiatric hospital admissions for AN did not differ between Netherlands Antilles immigrants and native Dutch,[79] suggesting that exposure to the Western beauty ideal is a risk factor for the development of AN, possibly in interaction with migration-related stress. A

similar finding for risk of BED among Mexican-American immigrants was found by Swanson and colleagues: in their study of a sample of people of Mexican origin in Mexico and the US, migration from Mexico to the US was associated with an increased risk of BED.[80] A recent study comparing prevalences of eating disorders across ethnic groups in the US reported similar prevalences of AN and BED among non-Latino whites, Latinos, Asians and African Americans. BN was more prevalent among Latinos and African Americans than among non-Latino whites.[48]

CONCLUSIONS

AN is relatively common among young women. While the overall incidence rate remained stable over the past decades, there has been an increase in the high-risk group of 15-19-year-old girls. It is unclear whether this reflects earlier detection of AN cases or an earlier age at onset. The occurrence of BN might have decreased since the early nineties of the last century. All eating disorders have an elevated mortality risk; AN the most striking. Compared with the other eating disorders, BED is more common among males and older individuals.

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Chapter 3

Three decades of eating disorders in Dutch primary care: decreasing incidence of bulimia nervosa but not of anorexia nervosa

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ABSTRACT

Background

Whether the incidence of eating disorders in Western, industrialized countries has changed over time has been the subject of much debate. The purpose of this primary-care study was to examine changes in the incidence of eating disorders in the Netherlands during the 1980s, 1990s and 2000s.

Method

A nationwide network of general practitioners (GPs), serving a representative sample (~1%) of the total Dutch population, recorded newly diagnosed patients with anorexia nervosa (AN) and bulimia nervosa (BN) in their practice during 1985-1989, 1995-1999, and 2005-2009. GPs are key players in the Dutch health care system, as their written referral is mandatory in order to get access to specialized (mental) health care, covered by health insurance. Health insurance is virtually universal in the Netherlands (99% of the population). A substantial number of GPs participated in all three study periods, during which the same case identification criteria were used and the same psychiatrist was responsible for making the final diagnoses. Incidence rates were calculated and for comparison between periods, incidence rate ratios.

Results

The overall incidence rate of BN decreased significantly in the past three decades (from 8.6 per 100,000 person-years in 1985-1989 to 6.1 in 1995-1999, and 3.2 in 2005-2009). The overall incidence of AN remained fairly stable during three decades, i.e. 7.4 per 100,000 person-years in 1985-1989, 7.8 in 1995-1999, and 6.0 in 2005-2009.

Conclusions

The incidence rate of BN decreased significantly over the past three decades, while the overall incidence rate of AN remained stable.

INTRODUCTION

Anorexia nervosa (AN) and bulimia nervosa (BN) are severe mental disorders with high mortality rates.[1,2] The etiology is still largely unknown.[3,4] Incidence studies examining secular trends enhance our understanding of how eating disorders develop, because changes in incidence over time may uncover risk factors. In developing countries, for example, increasing industrialization, urbanization and globalization are associated with an increase in eating disorders.[5] Whether the incidence of eating disorders in Western, industrialized countries has changed over time has been the subject of much debate.[6,7]

For AN, reports of an 'epidemic'[8] have been downsized to 'a modest increase in AN incidence over the 20th century',[9] by the identification of methodological confounders in long-term incidence studies such as variations in registration policy, diagnostic criteria, detection methods, and the availability of services; demographic differences between the populations; and faulty inclusion of readmissions.[7,10]

Less is known about secular trends in BN incidence. The diagnosis 'bulimia' was only introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) in 1980, and since 1970, few incidence studies have been conducted.[7,9] Most studies suggest an increase in BN incidence that reached a peak in the mid-1990s,[9,11-13] after which in UK primary care the incidence declined and then stabilized since 2000.[11,12] The observed increase might stem from increased recognition and help-seeking behavior of cases of a previously undefined disorder, instead of a true increase in incidence.[14] In a study comparing incidence rates of BN in Dutch primary care between 1985-1989 and 1995-1999, a decreasing trend was observed.[15] In line with this finding are the results from a prevalence study among college students in three periods,[16] which reported a lower BN point prevalence among women in 1992 and 2002 compared to 1982. In another US study among female students the point prevalence of probable cases of BN remained relatively stable between 1990 and 2004.[17]

In the present study, we want to enhance the limited knowledge on time trends in AN and BN, especially in the 21st century, by examining the incidence of AN and BN in Dutch primary care in 2005-2009, using the same methodology as our previous studies covering 1985-1989 and 1995-1999.[15,18-20] Data from general practitioners (GPs), serving a representative sample of the total Dutch population and instructed to detect eating disorders, were used, and diagnoses were established by systematic assessment of DSM-IV criteria. Time trends over three decades are reported.

METHOD

Sample

Since 1970, GPs participating in the sentinel practices of NIVEL Primary Care Database have continuously registered morbidity among their patients. Participating GPs were either recruited or selected (after application) based on the region and population density of their practice in order to ensure national representation. The GPs in the network weekly assessed and delivered data with regard to certain illnesses, events and procedures in general practice. Besides collection of regular weekly data, the participating GPs also provided annual data on relatively uncommon diseases, disorders and occurrences, such as eating disorders.[21] All these activities took about an hour per week for both the GP and the practice assistant. As compensation for their time investment participating GPs received an annual reimbursement.

The GPs had on average 148,326 patients in their practices during 1985-1989; 149,797 patients during 1995-1999, and 135,854 patients during 2005-2009. These patients were representative of the total Dutch population with respect to gender, age, regional distribution and population density, and covered about 1.0% of the Dutch population during 1985-1989 and 1995-1999, and 0.8% during 2005-2009. For example: in 2005-2009, 50.5% of the Dutch population was female; in the same period the total percentage of female patients registered in the sentinel practices was 50.7%. All 5-year age categories in the Dutch population were represented by 0.8 % or 0.9% in the population of the sentinel practices in 2005-2009.[21-25]

GPs in the Netherlands play a central role in the health care system and function as 'gatekeepers' to specialized care. Even if an individual contacts a medical specialist directly, the GP is always notified because health care insurers do not compensate for specialized health care costs without a written referral from the GP. Health insurance is virtually universal in the Netherlands: until 2006 health insurance through so-called 'sickness funds' was compulsory for low-income groups, while higher-income groups relied on private insurance. Since 2006 basic health insurance is obligatory for all Dutch residents, regardless of income. In 2008, 99% of the Dutch population thus had health insurance.[26]

During three periods, i.e. 1985-1989 (P1), 1995-1999 (P2), and 2005-2009 (P3), the participating GPs (n=82; average per period calculated over the three study periods; total n=164) registered the number of eating disorder patients in their practices. The P1 and P2 samples have been described extensively elsewhere.[15,19]

Procedure

Every year, the participating GPs received detailed information on eating disorders by means of a circular and at meetings convened for the purpose. With the use of case identification criteria described in Appendix 3.1, the GPs considered whether each patient who consulted them might be suffering from AN or BN. Sometimes the GP was alerted to the possibility of an eating disorder by other health care workers or worried relatives. To ensure consistency, the same case identification criteria were used in all three study periods and the same information on eating disorders was provided to the GPs. For each possible eating disorder patient, the GP completed an information sheet regarding eating disorder symptoms, height, weight, co-morbidity, and information on referral to specialized health care. The date of first detection by a health care professional (including, but not limited to the GP) was also noted. As in previous periods, registration forms of 2 years after the study period (2010 and 2011) were additionally screened for incident cases newly detected between 2005 and 2009, but not reported during that period. The research team made DSM-IV diagnoses of AN and BN on the basis of the information provided by the GPs. If necessary, the GP was contacted to provide additional information. During all three study periods the same psychiatrist (author H.W.H.) was responsible for scrutinizing the records from the GPs and making the final diagnoses, in order to ensure consistency in the systematic assessment of DSM-IV-criteria of AN and BN. There was a change from DSM-III-R to DSM-IV during the study periods, with more stringent criteria for BN in DSM-IV, while criteria for AN remained the same over time. To eliminate bias by this transition, the GP records of all possible cases from the first study period, which had originally been assessed with DSM-III-R criteria, were re-evaluated according to DSM-IV criteria.[15,19]

Statistical analysis

The incidence rate was defined as the number of new cases in primary care per year. It was based on the time when the eating disorder was detected, because it was unfeasible to assess precisely when the eating disorder began. Only patients with an eating disorder detected during 1985-1989, 1995-1999 and 2005-2009 were considered incident cases. The incidence rate was calculated by dividing the number of detected incident cases by the number of person-years in the Dutch sentinel practices. Person-years and number of cases per age group reported here differ slightly from those previously reported,[15] due to *post-hoc* updated information on the participating GPs and the registered cases. Incidence rates and their Poisson exact 95% confidence intervals (CIs) were calculated for the total population, for

females overall, and for females per 5-year age category. To determine if differences in incidence rates over time were statistically significant, incidence rate ratios (IRRs) were computed by dividing the incidence of P3 by the incidence of P1 ($IRR_{P_3\text{-}P_1}$), and P2 ($IRR_{P_3\text{-}P_2}$). We used STATA SE13[27] for the calculations of incidence rates and IRRs.

Ethical standards

The study was carried out according to the precepts of the Helsinki Declaration, Dutch legislation on privacy and the regulations of the Dutch Data Protection Authority. According to Dutch legislation, approval by a Medical Ethics Committee was not obligatory for this study, as the study used only anonymous data. In addition, the participants were not submitted to any medical interventions other than standard practice.

RESULTS

Anorexia nervosa

During 2005-2009, 41 patients (all female) were first diagnosed with AN, yielding an overall incidence rate of 6.0 per 100,000 person-years (Table 3.1, 95% CI: 4.3–8.1), which did not differ significantly from the overall rate in P1 and in P2 ($IRR_{P_3\text{-}P_1}$=0.8, 95% CI: 0.5–1.2; $IRR_{P_3\text{-}P_2}$=0.8, 95% CI: 0.5–1.1). The mean age at detection was 23.4 years (standard deviation (SD)=11.3, median 19.7 years, range 12.7-62.4 years), which did not differ significantly from the mean ages at P1 and P2 (F=0.4, df=2, p=0.7; for P1 and P2 see van Son et al.[15]).

Considering that the overall incidence rate was stable over time, and that the number of male incident AN cases was small in P1 and P2, it is not surprising that the incidence rate of AN in females remained stable as well ($IRR_{P_3\text{-}P_1}$=0.9, 95% CI: 0.6–1.3; $IRR_{P_3\text{–}P_2}$=0.8, 95% CI: 0.5–1.2). In all three study periods, the age-specific incidence was highest in the 15-19 years age group. The incidence rate among females aged 15-19 years initially increased between P1 and P2 ($IRR_{P_2\text{–}P_1}$=2.0, 95% CI: 1.1-3.7), but remained stable thereafter ($IRR_{P_3\text{-}P_2}$=0.9, 95% CI: 0.5–1.5).

Bulimia nervosa

During 2005-2009, 22 patients (including two men, aged 20 and 22 years, respectively, at detection) were first diagnosed with BN, yielding an overall incidence rate of 3.2 per 100,000 person-years (Table 3.2, 95% CI: 2.0–4.9). This rate differed

significantly from the overall incidence rate in P1 and in P2 (IRR_{P3-P1}=0.4, 95% CI: 0.2–0.6; IRR_{P3-P2}=0.5, 95% CI: 0.3–0.9). The mean age at detection was 24.8 years (SD=7.5, median 21.6 years, range 15.0-42.3 years), which did not differ significantly from the mean ages at P1 and P2 (F=0.9, df=2, p=0.4; for P1 and P2 see van Son et al.[15]).

The female incidence rate of BN was nearly three times higher in P1 than in P3 (IRR_{P3-P1}=0.3, 95% CI: 0.2–0.6; IRR_{P3-P2}=0.5, 95% CI: 0.3–0.8). During P1, the highest age-specific incidence was in the 25-29 years age group; during both P2 and P3, the age-specific incidences were highest in the 15-19 years age group.

DISCUSSION

In this Dutch primary-care study examining the incidence of AN and BN in the 1980s, 1990s and 2000s, the overall incidence of BN decreased significantly over the past three decades, while the incidence of AN remained fairly stable. The notion of the past century that BN is more common than AN[28,29] is therefore no longer valid. In the first decade of this century, the prominence of BN over AN seems to have disappeared (relative risk BN:AN=0.5, 95% CI: 0.3-0.9). Supporting evidence comes from a Finnish population cohort of female twins born during 1975-1979, in which the lifetime prevalence of AN was higher than that of BN (2.2% vs. 1.7%, respectively), and incidence rates among the high-risk group of females aged 15-19 years were comparable.[30,31]

AN is considered to be less prone to sociocultural influences than BN.[9,20] Historical, cross-cultural and biological evidence indicates that an AN-like syndrome has been existing across time, cultures,[9] and species.[32,33] Throughout the past centuries and across cultures, different motivations for the restriction of food have been ascribed to the patients, both by themselves and by their environment, such as religious motives (so called holy anorexia or anorexia mirabilis), digestive discomfort and weight concerns.[9] These motivations, however, impress more as a culturally meaningful attempt to understand AN than to have causal connotations.[9] AN may be seen as the formation of 'a well-ingrained and maladaptive habit'.[4]

For BN on the other hand, weight concerns and a desire to be slim seem crucial motivations to develop the disorder;[9] cognitive prerequisites impossible to create in animal models.[32] No historical or cross-cultural evidence of a binge-purge syndrome resembling BN exists outside the relatively new Western context of a culture that values and promotes the thin-body ideal.[9] Gaining more and more clinical

Table 3.1 Incidence anorexia nervosa per 100,000 person-years

Study period		**1985–1989**				**1995–1999**				**2005–2009**		
Females	**N**	**PY**	**IR**	**95% CI**	**N**	**PY**	**IR**	**95% CI**	**N**	**PY**	**IR**	**95% CI**
Age range, yr												
05–09	0	21,649	-	-	1	22,334	4.5	0.1–25.0	0	20,739	-	-
10–14	2	23,245	8.6	1.0–31.1	4	21,862	18.3	5.0–46.9	4	20,352	19.7	5.4–50.3
15–19	17	30,155	56.4	32.8–90.3	25	22,097	113.1	73.2–167.0	20	20,622	97.0	59.2–149.8
20–24	13	32,900	39.5	21.0–67.6	10	28,275	35.4	17.0–65.0	7	20,252	34.6	13.9–71.2
25–29	13	31,700	41.0	21.8–70.1	9	33,493	26.9	12.3–51.0	3	20,839	14.4	3.0–42.1
30–34	2	29,300	6.8	0.8–24.7	1	31,752	3.1	0.1–17.6	1	22,923	4.4	0.1–24.3
35–64	3	133,055	2.3	0.5–6.6	8	142,504	5.6	2.4–11.1	6	145,577	4.1	1.5–9.0
Overall females	50	373,975	13.4	9.9–17.6	58	381,399	15.2	11.6–19.7	41	347,764	11.8	8.5–16.0
Overall males + females	55	740,091	7.4	5.6–9.7	59	752,117	7.8	6.0–10.1	41	684,860	6.0	4.3–8.1

N=number of cases; PY=person-years; IR=incidence rate; CI=confidence interval

Table 3.2 Incidence bulimia nervosa per 100,000 person-years

Study period	1985–1989				1995–1999				2005–2009			
Females	**N**	**PY**	**IR**	**95% CI**	**N**	**PY**	**IR**	**95% CI**	**N**	**PY**	**IR**	**95% CI**
Age range, yr												
05–09	0	21,649	-	-	0	22,334	-	-	0	20,739	-	-
10–14	1	23,245	4.3	0.1–24.0	0	21,862	-	-	1	20,352	4.9	0.1–27.4
15–19	9	30,155	29.8	13.7–56.7	9	22,097	40.7	18.6–77.3	8	20,622	38.8	16.8–76.4
20–24	15	32,900	45.6	25.5–75.2	11	28,275	38.9	19.4–69.6	1	20,252	4.9	0.1–27.5
25–29	19	31,700	59.9	36.1–93.6	10	33,493	29.9	14.3–54.9	2	20,839	9.6	1.2–34.7
30–34	4	29,300	13.7	3.7–35.0	5	31,752	15.7	5.1–36.8	7	22,923	30.5	12.3–62.9
35–64	14	133,055	10.5	5.8–17.7	10	142,504	7.0	3.4–12.9	1	145,577	0.7	0.0–3.8
Overall females	62	373,975	16.6	12.7–21.3	45	381,399	11.8	8.6–15.8	20	347,764	5.8	3.5–8.9
Overall males + females	64	740,091	8.6	6.7–11.0	46	752,117	6.1	4.5–8.2	22	684,860	3.2	2.0–4.9

N=number of cases; PY=person-years; IR=incidence rate; CI=confidence interval

attention in the 1970s,[8] it was not until 1979 that the term 'bulimia nervosa' was coined by Gerard Russell in his influential paper describing several eating disorder cases with the binge-purge syndrome.[34] Keel & Klump[9] therefore conclude that BN is a culture-bound syndrome, while AN is not.

Which sociocultural developments in the last three decades might have caused the decreasing incidence of BN? Keel and colleagues[16] conducted a prevalence study among college students in three periods, and found a lower point prevalence of BN in 1992 and 2002 compared to 1982. They suggested that, by normalizing being overweight, a secular trend of an increasing body mass index (BMI) of the general population[35] might reduce the risk for developing BN. In the United States, the prevalence of obesity in both children and adults has stabilized in the first decade of the new millennium to around 17% and 35%, respectively.[36,37] Obesity is less common in the Netherlands than in the United States; the prevalence in adults, however, has risen steadily from 5% in 1985-1989, via 8% in 1995-1999, to 12% in 2005-2009 (http://statline.cbs.nl/statweb/). More specifically, the mean BMI of 20- to 29-year-old Dutch women increased significantly by 1.7 kg/m^2 during 1981-2004; a larger increase than for the total adult population (1.0 kg/m^2; men and women aged 20-69 years).[38] In exactly that group - women aged 20-29 years - incidence rates of BN plummeted between the study periods 1985-1989 and 2005-2009 ($IRR_{P_3\text{-}P_1}$=0.1, 95% CI: 0.0-0.5).

In a changing weight landscape, where a fuller-figured body is the norm, there might be less pressure to aggressively counteract the effects of binge eating by means of purging. Indeed, the declining rates of BN over time run parallel with the recognition of binge-eating disorder (BED) as a 'new' eating disorder diagnosis. BED was introduced in the DSM-IV in 1994 as a criteria set provided for further study, classified in the residual category 'eating disorder not otherwise specified' (EDNOS). In the latest edition of the DSM, DSM-5, BED is formally recognized as one of the specified eating disorders. BED and EDNOS were not covered in our study, and data on time trends in the occurrence of isolated binge eating in the Netherlands are not available. Circumstantial evidence comes from a recent community study of Dutch 20-year-olds, in which a relatively low lifetime prevalence of 0.8% for DSM-5 BN was found among young women, while the prevalence of BED was relatively high (2.3%).[39] A related but different perspective on the relationship between increased BMI of the population and binge eating is provided by a South-Australian community study[40] in which the point prevalence of disordered eating behaviors, including binge eating, doubled between 1995 and 2005, in parallel with a higher mean BMI of the sample. The authors argue that increased public

concern over obesity rates may lead to an increase in (unhealthy) weight control behaviors, with binge eating as a consequence of dietary restriction.[40]

Eating disorders in general received a fair amount of attention from both the scientific field[41] and the media from the nineties onwards.[42] Press coverage paid more attention to the (detrimental) medical consequences of eating disorders,[42] and prevention interventions were developed and tested, with variable success.[43] In the Netherlands, no national program on the prevention of eating disorders exists, and though some preventive activities were performed in the 2000s, those efforts reached only a minority of people.[44] However, eating disorders gained increased attention of Dutch policy makers: in 1998 the Steering committee Eating disorders Netherlands (SEN), a commission appointed by the Ministry of Health, Welfare and Sport, published a report with recommendations on specialized care for eating disorder patients. The first and only Dutch guidelines for the diagnosis and treatment of eating disorders were published in 2006.[45] These developments have not only lifted some of the taboo of - especially - BN, an eating disorder particularly surrounded by shame and secrecy, but also made clinicians, parents and teachers increasingly aware of the signs and symptoms of eating disorders. This may have resulted in early 'coming out' and recognition of BN patients and thus in earlier interventions, which could be reflected in a decreased peak age of incidence between the 1980s and the 1990s-2000s.

Although for both AN and BN mean age at detection did not differ between the three study periods, the proportion of patients aged <19 years increased over time. Whether this only reflects earlier detection or also earlier age at onset in younger generations is unclear.[46] In 2005-2009, virtually no new cases of BN were found in women aged 35-64 years. Beside the aforementioned hypotheses applying to the decreased incidence of BN in general, two additional explanations may hold for the decline in this age group in particular: first, the influx of more longstanding cases - previously undetected, but recognized after increased public and clinical awareness - coming to a halt.[14] Second, though recent evidence indicates that the menopausal transition poses increased risk for the development of eating disorders in middle-aged women, EDNOS seems more preponderant than either AN or BN in this age group.[47]

The majority of BN patients do not enter the (mental) health care system.[6,31] An additional reason for the decreasing BN trend in primary care may be the surge of alternative sources of help, such as self-help books and the internet during the 1990s.[11,42] The availability of self-help treatment may have helped many individuals to overcome their eating problems by themselves, and thus have prevented a

chronic course or the transition of subclinical symptoms into a clinical eating disorder.

The rapid expansion of internet availability in the Western world was a major sociocultural development in the 2000s. The Netherlands are in the lead when it comes to internet use: about 45% of Dutch citizens used the internet in 2000, to over 90% in 2010 (http://ec.europa.eu/eurostat/, http://statline.cbs.nl/statweb/). The influence of internet on eating disorders is a double-edged sword: on the one hand, pro-eating disorder websites exert a negative influence on eating disorders by promoting and reinforcing disordered eating behaviors;[48] on the other hand, barriers to treatment such as shame and fear of stigmatization are lowered by the anonymity of the medium, which makes internet a promising vehicle of delivering treatment for eating disorders.[49]

Strengths and limitations

Our study is the first that was specifically designed to examine secular trends by its focus on new rather than on prevalent cases of AN and BN. Another strength is that a large study population representative of the total (Dutch) population was investigated.

A potential challenge for studies covering three decades is that both case identification and case definition may have changed over time. Regarding case identification, in all three study periods the same criteria for case identification (see Appendix 3.1) were used. Moreover, a substantial proportion of GPs participated during the entire 25-year course of the study: of the GPs participating in 1985-1989 (n=67), 64.2% participated again in the second study period, and 34.3% in all three study periods. Taking into account that the participating GPs were annually instructed and that eating disorders have gradually become a standard component of the education program of medical schools, one would expect GPs to have become more familiar with eating disorders over time. This would have resulted in an increase in detection rates of eating disorders rather than a decrease, which was indeed our expectation at the start of the second and third study periods.

With respect to case definition, consistency of classification was ensured by applying DSM-IV criteria to all GP records of possible cases; including, in retrospect, to the records of the first study period, which had originally been evaluated using DSM-III-R criteria.[15,19] Another potential bias is a change over time in the case definition process by the research clinicians. However, while some members of the research team have changed, the final diagnosis was made by the same psychiatrist (senior author H.W.H.) over the three study periods.

Unfortunately we could include only AN and BN, and not the DSM-IV residual category EDNOS. EDNOS is probably the largest group of eating disorders in the community.[39,50] It is a heterogeneous category, which makes it difficult to assess consistently. Also, when this longitudinal study was set up in 1984, only AN and BN were defined in then current DSM-III and the case identification criteria were developed accordingly. To ensure methodological consistency over the years, these case identification criteria were neither changed nor adapted to developments in the definition and classification of eating disorders. Thus, the case identification criteria precluded the complete and systematic registration of residual category eating disorders such as for example binge eating without purging (BED) and purging without binge eating (purging disorder[51]). It is only with the recent introduction of DSM-5, that the broad EDNOS category was redefined, including the official recognition of BED as a new, specified eating disorder.[52] It seems unlikely that subjects who would currently be classified as BED were included in the BN cases. The same case identification criteria were used over the three study periods, and these criteria explicitly stated that compensatory behavior should be present in the case of BN. Furthermore, the frequency of purging behavior - necessary to fulfill DSM-IV criteria - was checked by the researchers based on the information provided by the GPs. Another limitation is that this study was conducted at the primary care level, so the reported incidence rates should be regarded as minimum estimates of the true rates in the community. Some underreporting cannot be excluded in this surveillance, which is integrated in routine medical care.

CONCLUSION

In conclusion, the incidence rate of BN decreased significantly over the past three decades, while the incidence rate of AN remained stable. Possible explanations for the decreased incidence of BN over time may be found in a sensitivity of BN to sociocultural influences and developments in the last 25 years, such as the effects of increasing obesity rates, and a rise of prevention efforts and alternative sources of help, augmented by the rapid expansion of internet availability.

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APPENDIX 3.1

Criteria for case identification* of anorexia nervosa and bulimia nervosa

Anorexia nervosa

1. Intense fear of becoming obese, even when underweight
2. Disturbance in the way in which one's body weight, size, or shape is experienced, e.g., claiming to "feel fat", even when emaciated, believing that one area of the body is "too fat" even when obviously underweight
3. Refusal to maintain body weight over a minimal normal weight for age and height, e.g., weight loss leading to maintenance of body weight 15% below expected weight; failure to make expected weight gain during the period of growth, leading to body weight 15% below expected weight

Bulimia nervosa

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than 2 hours)
2. During the eating binges, a feeling of lack of control over the eating behavior
3. Regular self-induced vomiting, use of laxatives, or rigorous dieting or fasting in order to counteract the effects of binge eating

*Case definition (by the research team) was based on more strict DSM-IV criteria

Chapter 4

Epidemiology, course, and outcome of eating disorders

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ABSTRACT

Purpose of review

To review the recent literature about the epidemiology, course, and outcome of eating disorders in accordance with the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5).

Recent findings

The residual category 'eating disorder not otherwise specified' (EDNOS) was the most common DSM-IV eating disorder diagnosis in both clinical and community samples. Several studies have confirmed that the DSM-5 criteria for eating disorders effectively reduce the proportion of EDNOS diagnoses. The lifetime prevalence of DSM-5 anorexia nervosa among women might be up to 4%, and of bulimia nervosa 2%. In a cross-national survey, the average lifetime prevalence of binge-eating disorder (BED) was 2%. Both anorexia nervosa and bulimia nervosa are associated with increased mortality. Data on long-term outcome, including mortality, are limited for BED. Follow-up studies of BED are scarce; remission rates in randomized controlled trials ranged from 19% to 65% across studies. On a community level, 5-year recovery rates for DSM-5 anorexia nervosa and bulimia nervosa are 69% and 55%, respectively; little is known about the course and outcome of BED in the community.

Summary

Applying the DSM-5 criteria effectively reduces the frequency of the residual diagnosis EDNOS, by lowering the threshold for anorexia nervosa and bulimia nervosa, and adding BED as a specified eating disorder. Course and outcome studies of both anorexia nervosa and bulimia nervosa show that no significant differences exist between DSM-5 and DSM-IV definitions.

INTRODUCTION

Epidemiological studies provide information about the occurrence of disorders and trends in the frequency of disorders over time, which knowledge is crucial to unraveling the etiology of (mental) disorders and for the purpose of planning treatment services. Course and outcome studies also serve to inform patients, clinicians, and policy makers on the prognosis of disorders, including the duration, effectiveness, and costs of treatment.[1] This review aims to provide an overview of the recently published studies on the epidemiology, course, and outcome of eating disorders, including anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED), and the residual diagnosis 'eating disorder not otherwise specified' (EDNOS, renamed 'other specified feeding or eating disorder' (OSFED) in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-5)[2]. We will update the reviews on this subject published in recent years by Keel & Brown[1] and Smink and colleagues.[3] In the first part, we define and describe the incidence and prevalence - the basic epidemiological measures - for AN, BN, BED, and EDNOS / OSFED in accordance with DSM-5 criteria. In the second part, we review course and outcome studies regarding the three eating disorders specified in the DSM-5: AN, BN, and BED.

EPIDEMIOLOGY OF EATING DISORDERS

Incidence is the number of new cases of a disorder in a population divided by the total time experienced for the population followed.[4] For eating disorders, the incidence rate is commonly expressed per 100,000 persons per year (100,000 person-years).[3] Prevalence is the proportion of a population that has a disorder at a specific point or interval in time, for example, at a certain date (point prevalence), in a certain year (12-month prevalence; often used in the DSM-5), or at any point in life (lifetime prevalence).

The DSM-5, published in May 2013, includes a revised eating disorder section.[2] A major problem with diagnosing eating disorders using the fourth edition (DSM-IV)[5] was the observation that the residual diagnosis EDNOS was the most common diagnosis in both clinical and community samples.[3,6] In order to reduce the frequency of EDNOS, the thresholds for AN and BN were lowered and BED was added as a specified eating disorder.[2,7] The revised definitions of the diagnostic categories will thus alter the disorder frequencies reported earlier.[3] In this section,

the epidemiological studies specifically focusing on eating disorders according to – or in line with – the DSM-5 criteria are discussed. For a more comprehensive review of the incidence and prevalence of AN, BN, BED, and EDNOS according to the DSM-IV criteria, see our recent review.[3]

Anorexia nervosa

Incidence studies of eating disorders on a community level are scarce. In a community study of female Finnish twins of the 1975-1979 birth cohort,[8] the incidence rate of a broad definition of AN (which is close to the DSM-5 definition of AN) was 490 per 100,000 person-years among 15-19-year-old girls, an increase of 81.5% compared with the incidence rate of DSM-IV AN (270 per 100,000 person-years). In the same study, the lifetime prevalence of AN almost doubled when the broad definition was used: 4.2% (broad AN) versus 2.2% (DSM-IV AN), which is in line with other community studies among women.[9,10] Stice et al.[11] followed a relatively small sample of 496 adolescent girls and found a lifetime prevalence of 0.8% for DSM-5 AN by age 20 (n= only 4). In a Portuguese sample of female college and university students,[12] DSM-IV cases of eating disorders were reclassified under the DSM-5 criteria. The point prevalence of DSM-5 AN was 0.7% with a cutoff point of body mass index (BMI) less than 17.5 kg/m^2 and 1.0% with a cutoff point of BMI less than 18.5 kg/m^2, compared with 0.6% for DSM-IV AN. The 12-month prevalence among young women is approximately 0.4%.[2]

Bulimia nervosa

For BN, the incidence rate among 16-20-year-old women from the Finnish female twin cohort increased by 50% from 200 to 300 per 100,000 person-years when symptom frequency was relaxed to once a week, in accordance with the DSM-5 criteria.[13] Several community studies found that the prevalence of BN increased by 30% when DSM-5 criteria were used, leading to a point prevalence of 0.6% for DSM-5 BN among adolescent girls and young women,[12] and a lifetime prevalence of around 2% for women.[11,13,14] The 12-month prevalence of BN among young women is 1-1.5%.[2,3]

Binge-eating disorder

To our knowledge, no incidence studies on BED yet exist. The 12-month prevalence of BED among US adult women and men is 1.6 and 0.8%, respectively.[2] Several population studies among adults found only very small increases in the lifetime prevalence of BED under the DSM-5 criteria,[14,15] leading to an estimated 12-month

prevalence for DSM-5 BED of 1.7% in US women, with the 12-month prevalence of men remaining at 0.8%.[15] In the first cross-national community survey including low-income, middle-income, and high-income countries,[16] an average lifetime prevalence of 1.9% for BED was found across the surveys. Lifetime prevalence estimates ranged from less than 1% in several European countries to 2.6% in the USA and 4.7% in Brazil; the sample from the latter country being limited to one urbanized area (Sao Paulo). Binge frequency had to be at least twice a week (in line with DSM-IV criteria), with a minimum duration of the disorder of 3 months (DSM-5).[16]

Eating disorder not otherwise specified / other specified feeding or eating disorder

The residual diagnosis EDNOS is a heterogeneous category, with different operationalizations across studies. Accordingly, the reported prevalence rates vary considerably.[2,17-19] The DSM-5 specifies five disorders in the residual OSFED category: atypical AN, BN of low frequency and/or limited duration, BED of low frequency and/or limited duration, purging disorder, and night eating syndrome. Little epidemiological data exist for most of these conditions. In a large community cohort of US adolescent girls and young women, the prevalence of purging disorder was 2-2.5%.[19] In another sample of 496 US adolescent girls, the lifetime prevalence by age 20 was 2.8% for atypical AN, 4.4% for subtreshold BN, 3.6% for subtreshold BED, and 3.4% for purging disorder; these conditions accounted for 68.9% of all DSM-5 eating disorder diagnoses.[11] However, several studies have confirmed that the DSM-5 criteria for eating disorders effectively reduce the proportion of EDNOS diagnoses, although the magnitude of the reduction varies across the studies.[12,20-23]

COURSE AND OUTCOME OF EATING DISORDERS

In the last decade, comprehensive reviews on the course and outcome of AN and BN have been published,[1,24-27] though of course they are based on studies using DSM-IV or DSM-III-R diagnostic criteria. Less is known about the course and outcome of BED because of a lack of long-term follow-up studies.[1] As BED is newly recognized as an official diagnosis in the DSM-5, we will relatively pay more attention in this review to the course and outcome studies of BED.

Important parameters of good outcome are remission and recovery. On the other end of the spectrum, mortality is the most extreme parameter of poor outcome.

Because mortality is such a salient and unambiguous outcome of eating disorders, we will review the recent studies on mortality in AN, BN, and BED irrespective of which DSM edition diagnostic criteria were used.

Anorexia nervosa

The crude mortality rate (CMR) is the number of deaths within the study population over a specified period. The standardized mortality ratio (SMR) is the percentage of observed deaths in the study population (e.g., AN patients) divided by the percentage of expected deaths in the population of origin (e.g., all women between 15 and 45 years of age).[3,4,28,29] In a meta-analysis of excess mortality in the 1990s, AN was associated with the highest rate of mortality among all mental disorders.[3,30] In a more recent meta-analysis of 35 published studies describing mortality rates for AN, the CMR for AN was 5.1 deaths per 1,000 person-years and the overall SMR was 5.9 with a mean follow-up period of 14.2 years.[31] A common cause of death in AN is suicide.[31-33] According to two meta-analyses (covering largely the same studies), the suicide rate for AN was 1.3 per 1,000 person-years; thus, one in five AN patients who die have committed suicide.[31,32] Other common causes of death in AN are the direct consequences of starvation and alcohol-related diseases.[33-38] Two recent studies using the Swedish Hospital Discharge Register found that fecundity is reduced in both men and women with (a history of) AN, compared with the general population,[39] but that women with a history of AN who do have children (before or after hospital admission for AN) have a better prognosis in terms of mortality.[40] This seems a rather straightforward finding, as infertility (amenorrhea) was a diagnostic criterion in the earlier editions of the DSM and is regarded as an indicator of physiological dysfunction in the DSM-5.[2] However, in the population-based study of the female Finnish twin birth cohort, the 5-year clinical recovery rate did not differ between AN patients with and without amenorrhea, being 66.8 and 69.1%, respectively.[8] This finding was replicated in a prospective study of an outpatient sample treated with cognitive-behavioral therapy (CBT): at 6-year follow-up, the recovery rate for AN according to both DSM-5 and DSM-IV criteria was 52%.[41] These results provide additional evidence for the validity of the deletion of the amenorrhea criterion in terms of treatment outcome.[42] However, the majority of AN patients in the community are not detected by the health care system[8] or referred to mental health care.[3,43]

Bulimia nervosa

Mortality and suicide risk are elevated in BN as well, albeit not as marked as in AN.[31,33] According to a meta-analysis of 12 studies describing mortality rates for BN, CMR was 1.7 per 1,000 person-years and SMR was 1.9 for a mean follow-up duration of 9.7 years.[31] Another recent meta-analysis of the risk of suicide in eating disorders[32] reported a suicide rate of 0.3 per 1,000 person-years for BN; thus, as is the case for AN, about one in five deaths in BN are the result of suicide. Several studies[13,41] report that recovery rates are the same for BN according to the DSM-5 and DSM-IV criteria. In a study of a treatment sample receiving CBT, recovery rate was around 50% for both DSM-5 and DSM-IV BN after 6 years of follow-up. Patients were considered recovered when they no longer fulfilled the criteria of any eating disorder at follow-up.[41] In the community study of female Finnish twins,[13] 55% of patients with BN according to the DSM-5 criteria were recovered 5 years after the onset of the disorder (DSM-IV: 57%). In this study, a more stringent definition of recovery was used: the absence of bingeing and purging for at least one year prior to assessment. Fewer than a third of the cases had been detected by health care professionals.[13] Keel et al.[44] reported that 20-year remission rates in a sample of college students did not differ significantly between DSM-IV BN and related EDNOS, such as BN of lower frequency and/or limited duration, BED, and purging disorder, suggesting that thresholds that distinguish BN from related EDNOS may have poor predictive validity.

Binge-eating disorder

Data on the long-term outcome of BED, including mortality, are scarce. None of the included studies in the meta-analysis of mortality rates in EDNOS by Arcelus et al.[31] stated explicitly that BED was part of the EDNOS category.[3,31] In a sample of 68 female inpatients with BED, CMR was 2.9% after 12 years of follow-up, with a nonsignificant SMR of 2.3 (95% CI: 0.0-5.5).[45] In a meta-analysis of three studies, no suicide had occurred among 246 BED patients after a relatively short mean follow-up of 5.3 years.[32]

Most outcome data on BED are (derived from) randomized controlled trials (RCTs) comparing different forms of psychotherapy and behavioral weight loss (BWL) treatment in overweight or obese BED patients. Duration of follow-up across the studies ranges from 1 to 6 years.[46-53] These studies address several important issues and priorities in BED research, such as the lack of long-term outcome studies[46,48,52,53] and the identification of predictors and moderators of treatment outcome.[48,51,53] In an RCT comparing cognitive-behavioral therapy-guided self-help

(CBT gsh), interpersonal therapy (IPT), and BWL, at 2-year follow-up significantly higher remission rates for binge eating were reported for both CBT gsh and IPT (62 and 67%, respectively) than for BWL (43%).[48] In a study examining the long-term efficacy of CBT and IPT, similar remission rates of 52.0 and 76.7%, respectively (non-significant difference; mean 64.4%), were observed after a mean follow-up duration of almost 4 years. Over the course of follow-up, remission rates declined significantly in the CBT group.[52] The attenuated efficacy of CBT over time was also observed in another study: whereas CBT provided better outcomes at the end of treatment compared with BWL, after 6 years of follow-up (retention rate 65%), remission rates in both treatment arms were around 19% only.[53] An Italian study comparing individual and group CBT in a treatment-seeking sample of both BED and subthreshold BED patients reported a relatively low remission rate of 32% after 3 years of follow-up; subthreshold BED was defined by a minimum binge frequency of once a week (in line with the DSM-5 criteria), for at least 6 months (DSM-IV).[46]

BED is associated with overweight and obesity.[48] The psychological treatments of BED do not produce substantial weight loss, although the elimination of binge eating might protect against future weight gain.[48,52,54,55] BWL leads to short-term weight loss,[48,49,51,53,55] but long-term weight loss has yet to be demonstrated.[53,55] However, in morbidly obese BED patients undergoing bariatric surgery, after 1 year the same amount of (substantial) weight loss was achieved as in surgically treated patients without BED, and, compared with BED patients receiving BWL, there were similar improvements in eating behaviors and risk factors for cardiovascular diseases.[56] BED may confer a risk of components of the metabolic syndrome (a cluster of related risk factors for cardiovascular disease, including abdominal obesity, dyslipidemia, hypertension, and abnormal glucose metabolism),[16,57] over and above the risk attributable to obesity alone.[57] Despite the important findings of these studies and their methodological rigor, including accurate assessment of BED with valid and reliable diagnostic instruments, it is important to note that from an epidemiological point of view they provide a limited picture of the true outcome of BED, as a substantial proportion of the total population of BED patients are not obese and the majority of BED patients do not seek treatment.[16]

There is a lack of longitudinal community studies on BED in adults, but for adolescents some studies of interest have been published recently.[11,19,58] In a prospective study of 8,594 adolescent girls evaluating whether BED predicted the development of adverse outcomes,[19] girls with BED had a twofold risk of becoming overweight or obese, or developing high depressive symptoms compared with nondisordered

girls, but the risk of starting to binge drink or use drugs was not increased. In another study of the same cohort in which boys were also included, weekly binge eating predicted drug use as well.[58] The presence of BED was ascertained by means of self-report and not all DSM-diagnostic criteria of BED were systematically assessed before making the diagnosis, which might have led to some misclassification.[19,58] Finally, in a community study of a sample of 496 female adolescents followed over 8 years with yearly assessment of eating disorders by means of a semi-structured interview, 3% developed DSM-5 BED, with a 1-year remission rate of 93%.[11] This most likely reflects the typical remission-relapse course of mild, self-limiting forms of BED in the community, for which no treatment is sought.[11]

CONCLUSION

The DSM-5 criteria for eating disorders effectively reduce the frequency of the residual diagnosis EDNOS, by lowering the threshold for AN and BN, and adding BED as a specified eating disorder. Both AN and BN are associated with increased mortality; for BED, however, data on the long-term outcome, including mortality, are scarce. The course and outcome studies of both AN and BN show that no significant differences exist between DSM-5 and DSM-IV definitions. For BED, most outcome data are derived from RCTs, whereas less is known about BED on a community level.

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Chapter 5

Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents

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ABSTRACT

Objective

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes a considerably revised eating disorder section. The aim of this study was to establish the prevalence and severity of eating disorders based on the new DSM-5 criteria in a community cohort of adolescents.

Method

This study is part of TRAILS (TRacking Adolescents' Individual Lives Survey), a Dutch cohort study on mental health and social development from preadolescence into young adulthood. At baseline, the participants (n=2,230) were about 11 years old. Body mass index was measured at all four assessment waves. At age 19, the Composite International Diagnostic Interview was administered to 1,584 of the participants. A two-stage screening approach was used to estimate the prevalence of DSM-5 eating disorders. Adolescents at high risk for eating disorders (n=312) were selected for an additional interview administered by eating disorder experts.

Results

Of the high-risk group n=296 (95%) could be interviewed. Among the women, the lifetime prevalence of DSM-5 anorexia nervosa was 1.7%, of bulimia nervosa 0.8% and of binge-eating disorder 2.3%. Eating disorders were relatively rare among the men. The severity of most cases was mild to moderate and detection and treatment rates depended on the level of severity.

Discussion

The most common DSM-5 eating disorder diagnoses in adolescents in the community are anorexia nervosa and binge-eating disorder. Severity ratings for eating disorders seem valid in terms of both the distribution in the community and the correlation with detection and treatment by health care services.

INTRODUCTION

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) was released in May 2013.[1] It includes a significantly revised eating disorder section.[2] The fourth edition, DSM-IV,[3] specified only two eating disorders, anorexia nervosa (AN) and bulimia nervosa (BN). In DSM-5, the eating disorder section is renamed 'Feeding and Eating Disorders' and specifies three eating disorders: AN, BN, and binge-eating disorder (BED); and three feeding disorders: pica, rumination disorder (RD), and avoidant/restrictive food intake disorder (ARFID). The DSM-IV listed some of the feeding disorders in the 'Disorders Usually First Diagnosed in Infancy, Childhood or Adolescence' chapter.

A major goal of the DSM-5 is to reduce the frequency of the DSM-IV residual category diagnosis 'eating disorder not otherwise specified' (EDNOS), a heterogeneous and not well-defined group of eating disorders which includes partial forms of AN and BN, purging disorder, and BED. EDNOS is the most common diagnosis in clinical[4] and community samples of adolescents, accounting in the latter for around 80% of all eating disorder diagnoses,[5,6] with psychopathology and adverse consequences comparable to AN and BN.[7,8] To achieve the desired reduction of this residual category (renamed 'other specified feeding and eating disorder' (OSFED) in the DSM-5), the criteria for AN and BN have been broadened[9,10] and BED has been added as a specified eating disorder. OSFED in the DSM-5 includes atypical AN, BN of low frequency or limited duration, BED of low frequency or limited duration, purging disorder, and night eating syndrome. In addition, the DSM-5 also includes the residual diagnosis 'unspecified feeding and eating disorder' when insufficient information is available to assign a more specific diagnosis. Studies evaluating whether the DSM-5 criteria effectively reduce the frequency of the EDNOS diagnosis show mixed results.[11-16] They are limited to treatment-seeking samples[12,13,15,16] or based on the reanalysis of cases previously diagnosed under DSM-IV criteria.[14,15]

Another new feature in the DSM-5 is the introduction of a severity rating for disorders, ranging from mild to extreme, in order to help clinicians track a patient's progress. Key severity parameters have been defined. Clinicians can increase severity ratings based on the severity of other (undefined) symptoms and the degree of functional disability.[1] The validity of severity ratings has not yet been investigated.

Eating disorders are relatively common in adolescents.[7,17] Little is known about the prevalence of eating disorders when new DSM-5-criteria are applied. To our knowledge, no study has yet assessed the prevalence of DSM-5 eating disorders

in a community cohort of both female and male adolescents. This study aimed to establish the lifetime prevalence of DSM-5 eating disorders, including severity ratings, in a 10-year follow-up study of a Dutch community cohort of adolescents. The distributions of eating disorder diagnoses according to DSM-5 and DSM-IV criteria were compared. Last, the validity of DSM-5 severity ratings was evaluated by examining their distribution in the community and their correlation with detection and treatment rates by health care professionals.

METHOD

Study design

A two-stage screening approach was used to estimate the prevalence of DSM-5 eating disorders in a community sample of adolescents. This approach is a widely accepted procedure for the identification of prevalent cases. [5,18-21] In the first stage, a group at high risk for an eating disorder was identified (see below for a detailed description of the selection criteria); in the second stage a structured clinical interview by telephone was administered to this group by eating disorder experts, to validate and classify the reported symptoms according to DSM-5 and DSM-IV criteria.

Study population

This study is part of a large Dutch prospective cohort study (TRAILS: TRacking Adolescents' Individual Lives Survey), which follows a community sample from preadolescence into early adulthood. The cohort has been extensively described elsewhere.[22,23] In 2001, 2,230 children (mean age 11.1 years, SD=0.6) from the north of the Netherlands, selected through community registers and through their schools, were enrolled in the study. The sample includes predominantly white children from five municipalities in both urban and rural areas. Follow-up assessments took place bi- or triennially (second assessment wave: n=2,149, response 96.4%, mean age 13.6 years, SD=0.6; third assessment wave: n=1,816, response 81.4%, mean age 16.3 years, SD=0.7). The cohort completed the fourth assessment wave in 2010 (n=1,881, response 84.3%, mean age 19.1 years, SD=0.6). Informed consent was obtained from the parent(s)/guardian of the participants at the first, second, and third assessment waves, and at the fourth wave from the participants themselves. The Central Dutch Medical Ethics Committee has approved the study.

Selection high-risk group

Adolescents at high risk for eating disorders were selected using measures collected at the fourth assessment wave, which included a comprehensive self-report questionnaire on (mental) health and social functioning, height and weight assessment by means of a standardized measurement procedure, and the Composite International Diagnostic Interview (CIDI) version 3.0,[24] a structured diagnostic interview for assessing current and lifetime DSM-IV disorders, administered by trained lay interviewers. See Table 5.1 for number and age of the study population at each stage of the study.

Table 5.1 Number and age of the study population per study stage*

	Number of adolescents (% female)	Mean age (SD)
Baseline assessment wave	2,230 (50.8)	11.1 (0.6)
Fourth assessment wave		19.1 (0.6)
- Questionnaire** completed	1,714 (54.7)	
- CIDI and/or BMI** completed	1,597 (53.9)	
- CIDI completed	1,584 (54.0)	
- BMI measured	1,560 (53.7)	
High-risk group eating disorders	312 (56.7)	19.1 (0.6)
Diagnostic interview eating disorders completed	296 (56.8)	20.6 (0.6)
- Lifetime DSM–5 FED diagnosis***	62 (80.6)	
- Lifetime DSM–5 ED diagnosis***	58 (84.5)	
- Lifetime DSM-IV ED diagnosis***	45 (84.4)	

* This study is part of TRAILS (TRacking Adolescents' Individual Lives Survey), a cohort study on mental health and social development from preadolescence into young adulthood.

** Questionnaire: comprehensive inventory on (mental) health and social functioning; CIDI: Composite International Diagnostic Interview; BMI: body mass index.

*** FED: feeding and eating disorder; ED: eating disorder.

Selection criteria for the high-risk group were based on the core features of an eating disorder: low or high body mass index (BMI), binge eating, compensatory behaviors and a distorted body image.[3] These core features were operationalized into 11 selection criteria. Adolescents who met at least one of these criteria were defined as being at high risk. A total of 312 adolescents (177 women / 135 men) fulfilled one or more of the selection criteria; see Table 5.2 for a more detailed description.

Table 5.2 Selection criteria high-risk group for eating disorders and endorsed proportion in a community cohort of adolescents*

Criterion	Variables**	Endorsed* % of women / % of men
Underweight	BMI <18.5	4.6 / 7.2
Obesity	BMI >30	6.3 / 5.0
Binge eating	CIDI-EA16*** = yes & BMI >27	3.1 / 1.5
Binge eating + compensatory behaviors	CIDI-EA24 = yes	5.0 / 2.7
Binge eating + compensatory behaviors: Vomiting	CIDI-EA16 = yes & Vomiting = very true or often true	0.1 / 0.0
Binge eating + compensatory behaviors: Excessive exercise	CIDI-EA16 = yes & Exercise = more than 60 minutes per day	1.0 / 1.6
Binge eating + compensatory behaviors: Fasting	CIDI-EA16 = yes & Breakfast frequency = never OR less than once a week	1.4 / 2.6
Binge eating + compensatory behaviors: Fasting	CIDI-EA16 = yes & Snack frequency = never	0.2 / 0.1
Distorted body image	CIDI-EA1 = yes & 18.5< BMI <20	2.0 / 0.3
Distorted body image	Body perception = a little too fat OR much too fat & 18.5< BMI <20	3.3 / 0.0
Distorted body image	Dieting = yes & 18.5< BMI <20	0.7 / 0.1

* n=1,597 (861 women and 736 men with completed CIDI and/or measured BMI at the fourth assessment wave of the TRAILS study (mean age 19.1 years)). A total of 312 (20.6% of the women and 18.3% of the men) fulfilled one or more of the selection criteria.

** Explanation of variables: BMI: body mass index. CIDI-EA1, CIDI-EA16, CIDI-EA24: Composite International Diagnostic Interview version 3.0 - eating disorders section: items 1 (weight concerns), 16 (binge eating) and 24 (binge eating and compensatory behavior). Vomiting**:** Adult Self Report (ASR): item 56g.[48] Breakfast frequency, snack frequency, body perception, dieting: questions developed by TRAILS, fourth wave questionnaire.

*** In CIDI-EA16, the CIDI-interviewers were instructed to leave out the frequency and duration criterion (binge-eating episodes at least two times a week for a couple of months). If binge eating was present, the interviewers asked the subsequent questions, regardless of frequency and/or duration.

Diagnostic interview

The high-risk group was invited to participate in an interview by telephone, in which the eating disorders module of the Structured Clinical Interview for DSM disorders (SCID-I)[25,26] and parts of the Eating Disorder Examination (EDE)[27] were administered by either a resident in psychiatry or a psychologist, both clinically experienced in diagnosing eating disorders. The duration of the interview could take up to one hour, depending on the adolescent's answers. Of the 312 high-risk adolescents, 296 (a 94.9% response rate) were interviewed over the telephone. One adolescent declined to participate and 15 adolescents could not be reached.

The goals of the interviews by telephone were to obtain current and lifetime diagnoses of feeding and eating disorders according to the DSM-5 and DSM-IV criteria, to define age at onset of the disorder, and to establish whether clinical recovery had occurred and whether the disorder had been diagnosed and treated by a health care professional. Because the SCID-I only covers AN, BN, and EDNOS-BED, skip rules were changed or omitted and questions from the EDE were added in order to diagnose other forms of EDNOS and DSM-5 AN, BN, and BED as well. In order to diagnose the three feeding disorders pica, RD, and ARFID, as well as the disorders listed in the residual category OSFED, diagnostic questions were formulated based on the proposed DSM-5 criteria for these disorders (as temporarily published on www.dsm5.org, accessed December 1, 2010). In order to assess potential eating disorder symptoms that may be more particular to men (e.g., muscle dysmorphia and drive toward muscularity), but which are not well captured in the DSM-IV and SCID-I, we explicitly asked the men about their body image and related self-evaluation, and past and current eating and sporting behaviors. For both men and women, also self-reported highest, lowest, and current weight, and explanations for current and past weight status were assessed. The interviewer wrote a narrative of each interview and the first author (FS) made a provisional diagnosis, also taking into account relevant data from previous assessment waves. All possible and probable eating disorder cases were discussed in a consensus meeting with an eating disorder expert and member of the DSM-5 Eating Disorders Work Group (co-author HWH). In 10 cases, additional information was necessary to establish a definite diagnosis and the respondent was called back to answer additional questions. Possible cases of feeding disorders were discussed with R. Bryant-Waugh, an expert on feeding disorders, also a member of the DSM-5 Eating Disorders Work Group.

For 15 out of 16 non-responders to the telephone interview, data on the eating disorders section of the CIDI (including audio recordings for 13 adolescents) were available and examined to assess whether there was sufficient information to ascertain or rule out an eating disorder diagnosis. This procedure yielded four definite eating disorder cases (two BN and two OSFED). In seven of the non-responder cases an eating disorder was ruled out. There was insufficient information available for five non-responders and these were considered missing.

Definition of disorder

Lifetime diagnoses of feeding and eating disorders were established using the DSM-5 criteria for feeding and eating disorders and the DSM-IV criteria for eating

disorders. However, due to the design of this study the focus was specifically on eating disorders, and less on feeding disorders. The diagnosis of feeding disorders, which generally occur in early and middle childhood,[28] is hampered by recall problems on the part of the adolescents.

Severity of AN, BN, BED, and feeding disorders was rated from mild to extreme in accordance with the DSM-5. For AN, the minimum level of severity is based on BMI (for adults; for children and adolescents on BMI percentiles), and may be increased to reflect clinical symptoms, the degree of functional disability, and the need for supervision. For BN and BED, the minimum severity levels are based on the frequency of inappropriate compensatory behaviors and episodes of binge eating respectively, and may be increased to reflect other (undefined) symptoms and the degree of functional disability. For the residual categories OSFED and 'unspecified feeding and eating disorder' and the feeding disorders, an assessment of severity is based on the intensity of physiological, behavioral and social impact parameters, and course of illness. In four cases (two BN and two OSFED), insufficient information was available to evaluate severity; these cases were conservatively labeled as mild.

Age at onset was defined as the age at which the symptoms of the eating disorder first occurred as reported in the diagnostic interview. In order to be classified as clinically recovered, the adolescent had to be asymptomatic for at least one year, here defined as restoration of weight and menstruation (AN, ARFID), and the absence of bingeing and purging for at least one year.[19,20,29] The one-year asymptomatic interval was chosen based on the literature, showing that for example in the case of BN the likelihood of symptom recurrence only decreases sharply after one year of abstinence from bingeing and purging.[29] Adolescents who had received an eating disorder diagnosis by a health care professional were considered 'detected', regardless of whether the diagnosis was followed by treatment.

Lifetime and point prevalence per DSM-5 and DSM-IV diagnosis were calculated by dividing the identified number of cases by the total number of adolescents who completed the CIDI and/or had their BMI measured at the fourth assessment wave (n=1,597; 1.3% CIDI without BMI; 0.8% BMI without CIDI). Confidence intervals (95%) for lifetime and point prevalences were calculated using exact methods in the statistical program R. Diagnostic crossover is common in eating disorders.[30] Multiple lifetime diagnoses of eating disorders in one adolescent were only treated as separate eating disorders if there was an asymptomatic interval of at least one year between two subsequent eating disorder diagnoses. When an adolescent did not have a one-year asymptomatic interval in between two different diagnoses,

only the diagnosis highest in the hierarchy of eating disorders (AN-BN-BED-OSFED) was included in the calculation of lifetime prevalence. The point prevalences include all adolescents with a lifetime diagnosis without clinical recovery at the time of the diagnostic interview.

Fisher's exact test was used to examine correlations between severity and detection rate, treatment rate, clinical recovery rate, and ANOVA was used to compare mean age at onset between severity categories.

RESULTS

Lifetime DSM-5 eating disorders

A lifetime diagnosis of any DSM-5 eating disorder was established in 5.7% of the female (95% confidence interval (CI): 4.2-7.5) and in 1.2% of the male adolescents (95% CI: 0.6-2.3). Five adolescents had more than one lifetime eating disorder diagnosis; however, none of them had experienced a one-year asymptomatic interval between diagnoses, so all five were only counted once in the prevalences. See Table 5.3 for the number of cases per diagnosis and sex-specific lifetime and point prevalences.

Table 5.3 DSM-5 eating disorders in a community cohort of adolescents*

DSM–5 Eating disorder**	Number of cases (% female)	Lifetime prevalence % (95% CI)**		Point prevalence % (95% CI)	
		Women	Men	Women	Men
AN	16 (93.8)	1.7 (1.0–2.9)	0.1 (0.0–0.8)	1.2 (0.6–2.1)	0.1 (0.0–0.8)
BN	8 (87.5)	0.8 (0.3–1.7)	0.1 (0.0–0.8)	0.6 (0.2–1.3)	0.1 (0.0–0.8)
BED	25 (80.0)	2.3 (1.4–3.6)	0.7 (0.2–1.6)	1.6 (0.9–2.7)	0.3 (0.0–1.0)
OSFED	7 (71.4)	0.6 (0.2–1.3)	0.3 (0.0–1.0)	0.3 (0.1–1.0)	0
Unspecified FED	2 (100.0)	0.2 (0.0–0.8)	0	0	0
Any ED	58 (84.5)	5.7 (4.2–7.5)	1.2 (0.6–2.3)	3.7 (2.6–5.2)	0.5 (0.1–1.4)

* Total n=1,597 (53.9% female) with completed CIDI and/or measured BMI at the fourth assessment wave of the TRAILS study (mean age 19.1 years); mean age at diagnostic interview by eating disorder experts 20.6 years.

** AN: anorexia nervosa; BN: bulimia nervosa; BED: binge-eating disorder; OSFED: other specified feeding and eating disorder; FED: feeding and eating disorder; ED: eating disorder; 95% CI: 95% confidence interval.

The most common diagnoses were AN and BED among the female adolescents, and BED among the male adolescents. However, absolute numbers of cases were small, especially in men, resulting in wide and often overlapping confidence intervals of prevalence estimates. Of the AN cases, 43.8% was classified as restricting subtype and 56.2% as binge/purge subtype. Of the 25 BED cases, 15 (60.0%) were obese (BMI >30) at the fourth assessment wave (mean age 19.1 years). Of all adolescents who were obese at the fourth wave, 16.5% had a lifetime diagnosis of BED. A minority of cases (seven adolescents; 12.1% of all eating disorder diagnoses) fell into the residual category of OSFED (one subthreshold BN, two subthreshold BED, two purging disorder, two night eating syndrome). Two other female adolescents displayed significant symptoms of an eating disorder without matching any of the descriptions in the OSFED category. One of them had extreme weight loss without being underweight and without the typical physiological and psychiatric correlates of AN; the other displayed several disordered eating behaviors accompanied by a self-evaluation unduly influenced by body weight and shape. They were categorized as having an 'unspecified feeding and eating disorder' (3.4% of all eating disorder diagnoses). Mean age at onset (SD) was 15.1 (2.8) years for AN, 16.0 (1.9) years for BN and 13.9 (2.4) years for BED. AN had the highest detection (68.8%) and treatment rates (56.3%) (see Table 5.5). Clinical recovery rates at the time of the diagnostic interview were 31.3% for AN, 25.0% for BN, 36.0% for BED, and 57.1% for OSFED.

Four cases of feeding disorders (three ARFID and one pica) with onset in late childhood or adolescence were identified, constituting 6.5% of all lifetime DSM-5 feeding and eating disorder diagnoses (see Table 5.1).

Comparison of DSM-5 and DSM-IV eating disorder diagnoses

A lifetime diagnosis of DSM-IV eating disorders was established in 4.4% of the female (95% CI: 3.1-6.0) and 1.0% of the male adolescents (95% CI: 0.4-1.9). The most common diagnosis for both sexes was EDNOS. Among the female adolescents, the lifetime prevalence of DSM-IV AN was 1.2% (95% CI: 0.6-2.1), of BN 0.6% (95% CI: 0.2-1.3), and of EDNOS 2.7% (95% CI: 1.7-4.0). Among the male adolescents, the lifetime prevalence of EDNOS was 0.8% (95% CI: 0.3-1.8). Of the AN cases, 45.5% were classified as restricting subtype and 54.5% as binge/purge subtype. One BN patient exhibited only non-purging compensatory behavior.

Table 5.4 shows the distribution of eating disorder diagnoses according to DSM-5 and DSM-IV criteria. Male and female adolescents combined, the lifetime prevalence of any eating disorder increased with 28.9% under DSM-5 criteria, mainly

Table 5.4 Comparative distribution of lifetime eating disorder diagnoses in a community cohort of adolescents* using DSM-5 and DSM-IV criteria

	DSM-IV Eating disorder**			
DSM–5 Eating disorder**	**AN (n=11)**	**BN (n=5)**	**EDNOS (n=29)**	**No DSM-IV ED diagnosis (n=1,552)**
AN (n=16)	11	0	5	0
BN (n=8)	0	5	3	0
BED (n=25)	0	0	18	7
OSFED (n=7)	0	0	3	4
Unspecified FED (n=2)	0	0	0	2
No DSM–5 ED diagnosis (n=1,539)	0	0	0	1,539

* Total n=1,597 (53.9% female) with completed CIDI and/or measured BMI at the fourth assessment wave of the TRAILS study (mean age 19.1 years); mean age at diagnostic interview by eating disorder experts 20.6 years.

** AN: anorexia nervosa; BN: bulimia nervosa; BED: binge-eating disorder; OSFED: other specified feeding and eating disorder; FED: feeding and eating disorder; ED: eating disorder; EDNOS: eating disorder not otherwise specified.

attributable to the inclusion of cases of BED who were subthreshold according to DSM-IV. Compared to the DSM-IV, the female lifetime prevalence of DSM-5 AN increased by 50.0% and of DSM-5 BN by 40.0%. According to DSM-IV criteria, 64.4% of all diagnosed cases (male and female) fell into the residual category EDNOS, which dropped to 15.5% for the residual DSM-5 categories OSFED and 'unspecified feeding and eating disorder' combined.

Severity ratings

Table 5.5 shows the distribution of severity levels, mean severity and detection and treatment rates per DSM-5 feeding and eating disorder diagnosis, and the proportion of detected and treated cases per severity level. Most cases were mild to moderate. AN had the highest detection and treatment rates; BED and the residual categories OSFED and 'unspecified feeding and eating disorder' the lowest. We found a statistically significant association between severity and detection rate (Fisher's exact test, p= .02) and between severity and treatment rate (p= .002). There was no statistically significant association between severity and clinical recovery rate (38.7% for all feeding and eating disorder diagnoses; p= .79). Age at onset did not differ between severity categories (mean age 14.5 years, median 14.5 years, SD=2.6; F=0.84, df=3, p= .48).

Table 5.5 Severity, detection and treatment rates per lifetime DSM-5 feeding and eating disorder diagnosis in a community cohort of adolescents*

	Severity n (row %)				Mean severity	Detected	Treated
DSM-5 Feeding and eating disorder**	**Mild**	**Moderate**	**Severe**	**Extreme**	**X (SD)*****	**n (row %)**	**n (row %)**
AN (n=16)	5 (31.2)	4 (25.0)	4 (25.0)	3 (18.8)	2.3 (1.1)	11 (68.8)	9 (56.3)
BN (n=8)	5 (62.5)	1 (12.5)	1 (12.5)	1 (12.5)	1.8 (1.2)	5 (62.5)	3 (37.5)
BED (n=25)	12 (48.0)	10 (40.0)	2 (8.0)	1 (4.0)	1.7 (0.8)	5 (20.0)	3 (12.0)
OSFED (n=7)	7 (100.0)	0	0	0	1	1 (14.3)	1 (14.3)
ARFID (n=3)	1 (33.3)	0	2 (66.7)	0	2.3 (1.2)	2 (66.7)	2 (66.7)
Pica (n=1)	1 (100.0)	0	0	0	1	0	0
Unspecified FED (n=2)	2 (100.0)	0	0	0	1	0	0
Any FED (n=62)	33 (53.2)	15 (24.2)	9 (14.5)	5 (8.1)	1.8 (1.0)	24 (38.7)	18 (29.0)
Number of detected cases (n=24) **(% per severity category)**	8 (24.2)	6 (40.0)	6 (66.7)	4 (80.0)			
Number of treated cases (n=18) **(% per severity category)**	4 (12.1)	5 (33.3)	5 (55.6)	4 (80.0)			

* Total n=1,597 (53.9% female) with completed CIDI and/or measured BMI at the fourth assessment wave of the TRAILS study (mean age 19.1 years); mean age at diagnostic interview by eating disorder experts 20.6 years.

** AN: anorexia nervosa; BN: bulimia nervosa; BED: binge-eating disorder; OSFED: other specified feeding and eating disorder; ARFID: avoidant/restrictive food intake disorder; FED: feeding and eating disorder.

*** Severity rating: mild=1; moderate=2; severe=3; extreme=4.

DISCUSSION

To our knowledge, this is the first study to assess DSM-5 eating disorder diagnoses with severity ratings in a community sample of female and male adolescents. The most common diagnoses were AN and BED among the women and BED among the men. BN was rare in our sample. The severity of most cases was mild to moderate and detection and treatment rates depended on the level of severity. Finally, while the lifetime prevalence of any eating disorder increased under the application of DSM-5 criteria, this application appeared to be successful in reducing the size of the DSM-IV residual category EDNOS.

DSM-5 eating disorders

The observed lifetime prevalence of 1.7% for DSM-5 AN among the female adolescents in our sample is close to prevalences between 2% and 4% for AN found in previous community studies among adult women using broad definitions of the DSM-IV, which overlap with the DSM-5 criteria.[19,31,32]

Compared to studies among US adolescents,[33-35] eating disorders with predominantly purging behaviors, such as BN and OSFED-purging disorder, were rare in our sample and less common than AN and BED. Possible explanations include the relatively young age of our sample, who was still in the middle of the high-risk period for BN[36] and an overall decreasing trend in the occurrence of BN over time.[36,37] The prevalence of OSFED-purging disorder in our study may be underestimated because the selection criteria for the high-risk group of eating disorders did not include isolated purging behaviors without the presence of binge eating. Cultural explanations for differences in prevalence between our study and studies in the United States include differences in ethnic composition of the samples,[38] urbanization,[39] over-the-counter availability of purging medications (such as diuretics and laxatives), and household composition. Our sample is predominantly white, while other studies might have included a larger proportion of respondents from ethnic minority groups.

The observed lifetime prevalence for BED of 2.3% among the women is comparable to other studies among female adolescents.[34,35] Recent epidemiological studies among adolescents[33-35] indicate that BED is more common among adolescents than previously thought.[40] The majority of DSM-5 BED cases in our sample had mild to moderate severity ratings.

Although we diagnosed four cases of feeding disorders with onset in late childhood or adolescence, we expect the true prevalence to be higher due to our study

design, which is not optimal for detecting feeding disorders. Methodological problems include the selection criteria for the high-risk group that insufficiently capture feeding disorder symptoms, and recall problems of the adolescents. Parent interviews could provide a valuable contribution to the detection of feeding disorders.

Only one male case of AN was found, compared to 15 female cases. For BED, a male-female ratio of 1:3.4 was observed, quite similar to the ratio found in a large population sample of US adolescents.[33] However, due to small numbers of cases in our study, especially in men, male-female ratios should be interpreted with caution. For example: when taking into account (unrounded) confidence intervals, the male-female ratio of BED ranges from 1:1 to 1:16.

Comparison of DSM-5 and DSM-IV eating disorder diagnoses

The lifetime prevalence of any eating disorder for male and female adolescents combined increased by 28.9% under the DSM-5 criteria, mainly attributable to the inclusion of BED cases that were subthreshold according to DSM-IV criteria. Population studies among adults found small increases in the prevalence of BED under the DSM-5 criteria.[41,42] The validity of DSM-5 BED classification has been criticized for setting the threshold for a psychiatric disorder too low.[43] However, studies among adolescents indicate that binge eating with a frequency of at least once a week is a predictor for negative outcomes, such as the development of overweight/obesity and depressive symptoms and the onset of drug use.[34,44] The question of where to place the cut-off between problematic behavior and disorder remains though. Often, one of the features of a mental disorder is the presence of distress and/or functional disability. The 'distress criterion' (Criterion C in BED) needs to be met in order to establish a diagnosis of BED. The presence of distress in each possible case was extensively discussed in our diagnostic consensus meetings in order to make sure that a diagnosis of (subthreshold) BED was limited to adolescents who displayed the full syndrome and not only the symptom 'binge eating'.

In our sample, the lifetime prevalence of AN and BN among female adolescents increased by 50.0% and 40.0%, respectively, with the use of DSM-5 criteria. Earlier community studies have suggested that the lifetime prevalence of AN among women doubled when the amenorrhea criterion was dropped, in line with the DSM-5 criteria for AN.[19,31,32] In a community sample of female high school and university students, the point prevalence of AN increased by 61% when DSM-5 criteria were used.[14] Using the DSM-5 criteria and relaxing the requirement for

binge and compensation frequency from two to one per week, the prevalence of BN increased by 30% in several community studies.[14,20,42]

The DSM-5 criteria for eating disorders effectively reduced the contribution of the residual category, from 64.4% (DSM-IV EDNOS) to 15.5% (DSM-5 OSFED and 'unspecified feeding and eating disorder' combined). Other studies evaluating whether the DSM-5 criteria effectively reduce the proportion of the EDNOS category also show a reduction of the residual category,[11-16] however, none as large as the reduction in our sample.

Severity ratings

As was to be expected for a community cohort, the majority of feeding and eating disorder cases had mild to moderate severity. This finding, combined with the observation that a significant association exists between severity and the proportion of cases detected and treated by (mental) health care services, provides evidence that the DSM-5 severity ratings are valid. We found no difference in age at onset between severity categories, reflecting that age at onset is probably more disorder- than severity-dependent. Neither did we find a correlation between severity and the proportion of clinically recovered cases. This could be explained by the relatively young age of our sample, combined with a high threshold for clinical recovery. In other words: it might simply be too early to tell if severity correlates with clinical recovery rate. Moreover, cases that were labeled mild at the time of the diagnostic interview might just have been in the early stage of the disorder, and progress to a more severe level over time. In the next assessment wave of this cohort study we hope to examine how severity ratings develop over a longer follow-up period, and how they correlate with other measures, e.g., quality of life.

Strengths and limitations

Strengths of this study include its community-based design, including both female and male adolescents. Eating disorder experts administered diagnostic interviews and participated in diagnostic consensus meetings. Response rates were high, and we managed to confirm or rule out an eating disorder diagnosis for the majority of non-responders. Diagnoses were made on the basis of all available information, including data collected in previous assessment waves.

However, this study also has several limitations. First, possible selection bias of the study sample: non-responders at the fourth assessment wave (n=349; 15.6% of the baseline sample) were more likely to be male, and, compared to responders, they more often had divorced parents, a low educated mother, low family income,

low socioeconomic position, low academic achievement, and low peer status at the first assessment wave. Also, non-responders more often used tobacco and cannabis than responders.[45] In a previous study among US adolescents,[33] measures of socioeconomic status, such as parental education, household income and parental marital status were not significantly associated with any eating disorder presentation, while substance abuse was associated with BN and BED. We therefore believe that BN and BED may be slightly underestimated in our study.

Second, some limitations pertain to the first stage of case finding: the selection of the high-risk group for eating disorders. In order to fulfill the dimension 'binge eating', the adolescents had to answer yes to the item on binge eating in the CIDI-interview, in combination with having a BMI >27 at age 19. We decided on a minimum BMI in order to increase the specificity of the selection criterion and the likelihood of problematic behavior. We might have missed instances of BED in adolescents who had a BMI <27 at age 19, and were unselected for that reason. According to De Zwaan,[46] 50% of BED cases in community samples are overweight. Thus, our estimate of the prevalence of BED might double when normal-weight adolescents would have been included in the high-risk definition of binge eating. Our lifetime prevalence of BED has therefore to be regarded as a minimum estimate. Also, purging behaviors were assessed only in combination with binge eating, which might have led to an underestimation of OSFED-purging disorder.[47]

Third, a limitation of the second stage of the study - the diagnostic interview – is the fact that it was administered by telephone rather than in person, thereby excluding the interpretation of potentially important non-verbal information and the opportunity to measure current weight and height. However, a telephone interview was advantageous in that it required a smaller effort from the participant, and was more anonymous than a face-to-face interview would have been, resulting in seemingly frank answers on questions about shameful and secretive behaviors, such as binge eating. Moreover, weight and height had been measured in person at all assessment waves of the study. Another limitation pertains to determining age at onset of the eating disorder: because the development of an eating disorder is usually a gradual process, it is difficult to pinpoint the onset in time. Last, recall problems play a role, even in our relatively young study population.

CONCLUSIONS

AN and BED are the most common eating disorders among adolescents, especially the mild forms. In DSM-5, the proportion of diagnoses in the residual category EDNOS is effectively reduced. Severity ratings for eating and feeding disorders seem valid in terms of distribution in the community and correlation with detection and treatment by (mental) health care services.

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Chapter 6

Self-perceived and peer-perceived social status in early adolescence and risk of eating pathology in young adulthood

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In revision

ABSTRACT

Background

Self-perception of social status is associated with eating pathology, but little is known about the influence of social status as judged by others. The present study investigates whether self-perceived and peer-perceived social status among early adolescents are associated with eating pathology in young adulthood.

Methods

This study is part of TRAILS, a longitudinal cohort study on mental health and social development from preadolescence into adulthood. At a mean age of 11.1 years, the participants enrolled in the study and completed the Self-Perception Profile for Children, assessing children's general self-worth, and self-esteem regarding social acceptance, physical appearance and academic competence. At age 13.6, peer status among class mates was assessed on domains regarding affection (social acceptance) and achievement (physical attractiveness, academic competence and popularity) in a subsample of 1,007 participants. The Eating Disorder Diagnostic Scale was administered at age 22.3. The present study included peer-nominated participants for whom body mass index at age 11 and 13, and completed measures of self-perception and eating pathology at age 22 were available, resulting in a final subsample of 699 participants (57.8% female).

Results

Both self-perceived and peer-perceived physical attractiveness were inversely correlated with eating pathology at 22 years. Low peer popularity predicted eating pathology, but this effect disappeared when the other peer-status domains were controlled for. On the contrary, high social acceptance by peers emerged as a predictor of eating pathology only after adjustment for the other status domains.

Conclusions

In early adolescence, looking good – either in one's own eyes or in the eyes of peers – protects against eating pathology in young adulthood. However, being liked by peers might indicate a risk.

INTRODUCTION

Disordered eating behaviors and eating disorders usually develop in adolescence.[1] The adverse consequences on both mental and physical health are unequivocal.[2] Uncovering risk factors for eating pathology may offer clues for prevention. This study aims to disentangle the respective roles of self-perceived and peer-perceived social status in the risk of developing eating pathology.

Low self-esteem is a relatively well-established, albeit non-specific, risk factor for eating pathology.[3] Self-esteem develops in late childhood and early adolescence and is to a great extent shaped by two processes: social comparison with and social support by significant others such as peers.[4] Self-perceived low social status has been linked to a range of mental disorders,[5] including eating disorders (e.g., Troop et al.[6]). In contrast to self-perceived social status, little is known about social status as perceived by others and its potential role in the onset of eating pathology.

Social status can be divided into affection-related status reflecting social acceptance (who is liked), and achievement-related status reflecting the hierarchical structure of the group (who is best). Only a few studies examined the association between affection-related status and weight-related behaviors and cognitions. Graham et al.[7] found that adolescents who are well-liked by peers, as assessed with peer nominations, are more satisfied with their bodies. This is in line with a longitudinal study[8] showing that well-liked early adolescents had fewer weight-related behaviors and cognitions after eleven months than disliked adolescents. Lieberman et al.[9], on the other hand, found that girls who received more friendship nominations were *more* likely to exhibit disordered eating behaviors and displayed lower body esteem, while Wang and colleagues[10] found no association between adolescents' affection-related status and body size or dieting behavior.

Achievement-related status refers to popularity or social impact.[11] This 'social attention holding power' does not equate with social acceptance.[12] In a cross-sectional study among 17-year-old boys and girls, high peer popularity was associated with more dieting behaviors and a body shape that fits the current ideals for men (muscular) and women (thin) (Wang et al.).[10] Rancourt & Prinstein[8] found that highly popular adolescents were at greater risk of developing weight-related behaviors and cognitions than less popular peers. To our knowledge, there are no studies on eating disorder pathology in relation to other domains of achievement-related status, such as academic competence and physical attractiveness.[13] Research did show that obese adolescents are less popular and more often socially rejected, viewed as less attractive, and labelled as 'stupid' or 'lazy'.[14]

In the present study, we investigated associations between early adolescent self-esteem and classroom social status, and eating pathology in young adulthood. Two research questions were formulated: 1. Is self-esteem in early adolescence associated with eating pathology in young adulthood? 2. Is achievement-related (physical attractiveness, academic competence, popularity) and affection-related (social acceptance) classroom social status in early adolescence associated with eating pathology in young adulthood? We hypothesized that low self-esteem would increase risk of developing eating pathology later on. Furthermore, low status in affection-related domains was hypothesized to be a risk factor for the development of eating pathology, and high status a protective factor. Considering the unstable social hierarchy of early high school, both low and high status in achievement-related domains are inherently stressful and thus were expected to increase risk of eating pathology.

METHOD

Study population

This study used a subsample of the Dutch prospective cohort study TRAILS (TRacking Adolescents' Individual Lives Survey), which follows a community sample from early adolescence into young adulthood. The cohort has been extensively described elsewhere.[15] In 2001, 2,230 children (mean age 11.1 years, SD=0.6) from the north of the Netherlands, selected through community registers and through their schools, enrolled in the study. The sample includes predominantly Caucasian children from five municipalities in both urban and rural areas. Follow-up assessments took place bi- or triennially (second assessment wave (T2): n=2,149, response 96.4%, mean age 13.6 years, SD=0.5; third assessment wave (T3): n=1,816, response 81.4%, mean age 16.3 years, SD=0.7; fourth assessment wave (T4): n=1,881, response 84.3%, mean age 19.1 years, SD=0.6; and fifth assessment wave (T5): n=1,782, response 79.9%, mean age 22.3 years, SD=0.7). The proportion of female participants ranged from 50.8% (T1) to 52.7% (T5). Informed consent was obtained from the parent(s) or guardian of the participants at T1-T3, and at T4 and T5 from the participants themselves. The Central Committee on Research Involving Human Subjects approved the study.

At T2, classroom social status on several domains was assessed by means of peer nominations in classes with at least three TRAILS participants.[16] Peer nominations were obtained in 172 classes (72 first grade and 100 second grade of secondary edu-

cation) at 34 schools. The school classes were more or less equally distributed across educational levels: 60 low, 53 middle, and 59 high education. The mean number of students in each participating school class was 18.4 (SD=6.0, range 7-30). A total of 3,312 adolescents nominated their class mates, which yielded peer nominations for 1,007 TRAILS participants (51.3% female). The analyses in the present study included peer-nominated participants for whom adjusted body mass index (BMI; see Measures-*Eating pathology*) at T1 and T2, and completed measures of self-esteem (T1) and eating pathology (T5) were available, resulting in a final subsample of 699 participants (57.8% female). Compared to the rest of the total TRAILS cohort, this subsample had a lower adjusted BMI at T1 and T2, and slightly less eating pathology and higher self-esteem regarding academic performance, social acceptance and general self-worth at T1. Furthermore, they were somewhat younger at T2 and T5, more often nominated for good looks and being a good learner, and less often nominated for being popular at T2 (see Table S6.1 in Supplemental Tables).

Measures

Self-perception

The Self-Perception Profile for Children (SPPC)[17] was administered at T1. The SPPC assesses children's general feelings of self-worth and self-esteem in five specific domains (academic competence, social acceptance, athletic competence, physical appearance and behavioral conduct). Higher scores indicate higher self-esteem. The SPPC has been shown to have good reliability and validity.[18] For this study, we included SPPC domains that corresponded with the peer-status domains under study, that is, academic competence, social acceptance, physical appearance, and general self-worth. Cronbach's alpha ranged from .71 to .81 for these SPPC scales.

Peer status

Students could nominate their class mates on a total of eighteen topics, of which four on achievement-related and affection-related status were selected for this study. Achievement-related questions concerned physical attractiveness ('Who are good-looking?'), academic competence ('Who are good at learning?'), and popularity ('Whom do others want to be associated with?'). With regard to the popularity question, students were asked whom they thought *others* wanted to associate with, in order to disentangle personal preference from social impact.[16] Affection-related status was assessed by the question 'Which classmates do you like?'. The number

of received nominations was divided by the number of classmates, yielding a proportion score ranging from 0 (no nominations) to 1 (nominated by all classmates).

Eating pathology

Eating pathology was measured at T5 by means of the validated Dutch translation of the Eating Disorder Diagnostic Scale (EDDS), a 22-item self-report questionnaire that generates DSM-IV diagnoses of anorexia nervosa (AN), bulimia nervosa (BN) and binge-eating disorder (BED), and an overall symptom composite score.[19-21] For this study we only used the composite score, which was constructed by summing the raw items, excluding items regarding height, weight and use of birth control. It indicates the level of eating pathology; not the specific type of eating problems, and has been shown to have satisfactory internal consistency.[20]

To control for eating pathology at baseline, two sum scores – one for T1 and one for T2 – were constructed using measures collected at the first two assessment waves. Since no specific eating disorder diagnostic instrument was administered at T1 and T2, a proxy measure was created by means of indicators of eating pathology based on its core features: high or low BMI, binge eating, compensatory behaviors and distorted body image.[22] A more detailed description of the proxy measure is provided in Supplemental Information and in Table S6.2 in Supplemental Tables.

Statistical analysis

First, means of all variables used in the study were calculated per gender and differences between genders tested with t-tests. Second, for both genders, correlations between self-esteem variables, peer-status domains, T1 and T2 baseline eating pathology scores and T5 eating pathology were calculated. Correlation coefficients between .20 and .40 were considered weak, between .40 and .60 moderate, and higher than .60 strong.

Subsequently, a linear regression model was used to predict eating pathology at T5 by various domains of self-esteem at T1, both unadjusted and adjusted for baseline eating pathology at T1. Next, all self-esteem domains were adjusted for one another to assess the specific contribution of each domain. Subsequently, we predicted eating pathology at T5 by the peer-status variables assessed at T2, both unadjusted and adjusted for baseline eating pathology at T2, also by means of linear regression analyses. Again, the effect of peer status on later eating pathology was assessed for each individual peer-status domain separately, and for all peer-status domains adjusted for one another. Because achievement-related peer-status variables were hypothesized to have a curvilinear relationship with

eating pathology at T5, a quadratic term of each achievement-related peer-status variable (physical attractiveness, academic performance, popularity) was added to the regression model. Since the eating pathology score of T2 was based on more variables than the score of T1, the T2 models were also run while adjusting for a T2 eating pathology score that was based on the same variables as the T1 score, in order to ensure comparability of the two models. All results were adjusted for gender. To examine if gender moderated the results, we added an interaction term to the model for each predictor variable (predictor X gender), and tested whether this significantly increased the explained variance by means of an F-test. When the F-test was non-significant, the interaction term was dropped. The significance threshold was set at .05.

RESULTS

Descriptive statistics

Table 6.1 shows descriptive statistics of the variables used in this study. Regarding physical appearance and academic competence, girls displayed lower self-esteem, but were more often nominated for being beautiful and for being a good learner (difference marginally significant; $p = .06$) by their class mates than boys. Furthermore, females had significantly higher eating pathology scores at T2 and T5.

Bivariate associations

For both boys and girls, correlations between self-esteem domains were generally weak, except for the domains of general self-worth and physical appearance, which were strongly correlated, and general self-worth and social acceptance, which showed a moderate association (Table 6.2). Self-perception and peer-perception of social acceptance and academic performance were weakly correlated. No correlation between self-perception and peer-perception of physical appearance was found. Appearance-related self-esteem and general self-worth showed a weak negative correlation with eating pathology at T5. Regarding peer status, being nominated for good looks was moderately correlated with being liked and being popular in both boys and girls. In girls, higher peer popularity was associated with lower levels of eating pathology cross-sectionally.

Table 6.1 Means (SD) of self-esteem, peer status, eating pathology and body mass index, by gender

Self-esteem* (T1)**	**Females (n=404)**	**Males (n=295)**	**Difference *t*-test (*p*-value)**
Social acceptance	3.10 (0.56)	3.11 (0.54)	$t = -0.33$ (.74)
Physical appearance	3.08 (0.61)	3.24 (0.57)	$t = -3.67$ (< .001)
Academic performance	2.90 (0.49)	2.98 (0.50)	$t = -2.01$ (.045)
General	3.35 (0.52)	3.40 (0.50)	$t = -1.35$ (.18)
Peer status* (T2)**			
Social acceptance	0.57 (0.20)	0.56 (0.20)	$t = 0.62$ (.54)
Physical attractiveness	0.27 (0.23)	0.13 (0.14)	$t = 10.23$ (< .001)
Academic performance	0.35 (0.26)	0.31 (0.26)	$t = 1.89$ (.06)
Popularity	0.10 (0.12)	0.10 (0.13)	$t = 0.58$ (.57)
Eating pathology*			
T1	0.13 (0.38)	0.08 (0.31)	$t = 1.82$ (.07)
T2	0.26 (0.57)	0.18 (0.45)	$t = 2.11$ (.04)
T5**	13.28 (10.57)	5.48 (6.78)	$t = 11.86$ (< .001)
Adjusted BMI*			
T1	102.74 (15.84)	102.28 (13.62)	$t = 0.40$ (.69)
T2	101.61 (16.06)	100.24 (13.83)	$t = 1.18$ (.24)

* Self-esteem: Self Perception Profile for Children (SPPC)[17]; scores could range between 1 (low self-esteem) and 4 (high self-esteem). Peer status: number of received nominations by class mates divided by total number of class mates; scores could range between 0 (no nominations) and 1 (nominated by all class mates). Eating pathology at T1 and T2: a proxy measure was created by means of indicators of eating pathology based on its core features (high or low body mass index, binge eating, compensatory behavior and distorted body image). Endorsement of an indicator added one point to the sum score. Scores could range between 0 and 4 for T1, and between 0 and 7 for T2. Eating pathology at T5: symptom composite score of the Eating Disorder Diagnostic Scale (EDDS)[19,21]; scores could range between 0 and 113 for women, and between 0 and 109 for men. Adjusted BMI: adjusted body mass index (%) = (BMI / 50th percentile BMI for age and gender) x 100.

** T1: mean age 11.1 years; T2: mean age 13.6 years; T5: mean age 22.3 years.

Self-esteem and risk of eating pathology

Table 6.3 shows standardized regression coefficients of the four self-esteem domains and the four peer-status domains, both unadjusted and adjusted for baseline eating pathology (at T1 and T2, respectively) and for other domains of self-esteem and peer status. No moderating effect of gender on any of the self-esteem domains was found. Lower appearance-related self-esteem at 11 years predicted higher levels of eating pathology at 22 years, also after adjusting for baseline eating pathology and other self-esteem domains. Lower general self-worth predicted later eating pathology as well, but this effect disappeared when the other self-esteem domains were controlled for.

Table 6.2 Correlations between self-esteem domains, peer-status domains and eating pathology in girls (above the diagonal) and boys (below the diagonal)

	Self-esteem (T1)* **				**Peer status (T2)*** **				**Eating pathology**		
Self-esteem (T1)	Social acceptance	Physical appearance	Academic performance	General	Social acceptance	Physical attractiveness	Academic performance	Popularity	T1	T2	T5***
Social acceptance		.32**	.19**	.41**	.21**	.19**	.09	.14**	.02	-.02	-.08
Physical appearance	.33**		.16**	.68**	-.07	-.06	.12*	-.11*	-.02	-.05	-.20**
Academic performance	.30**	.24**		.26**	-.03	-.02	.28**	.01	-.04	.02	-.02
General	.41**	.72**	.33**		-.05	-.06	.15**	-.06	.04	.02	-.14**
Peer status (T2)											
Social acceptance	.23**	-.07	.09	.02		.58**	.03	.28**	-.05	-.05	.04
Physical attractiveness	.20**	-.11	.02	-.01	.47**		.05	.49**	-.11*	-.06	-.08
Academic performance	-.04	-.01	.12*	-.10	.08	.02		-.14**	-.04	.05	.04
Popularity	.11	-.02	-.01	-.05	.16**	.45**	-.03		-.05	-.15**	-.06
Eating pathology											
T1	-.03	-.09	.02	-.01	-.05	.04	.05	-.03		.35**	-.02
T2	-.02	-.15*	-.01	-.09	-.09	-.02	.02	-.05	.47**		-.01
T5	-.05	-.18**	.01	-.15*	.02	-.05	.00	-.04	.11	.07	

* $p < .05$

** $p < .01$

*** T1: first assessment wave of the TRAILS study, mean age 11.1 years; T2: second wave, mean age 13.6 years; T5: fifth wave, mean age 22.3 years.

Peer status and risk of eating pathology

Gender did not moderate the relationship between any of the peer-status domains and later eating pathology. Addition of a quadratic term of the achievement-related peer-status variables in the regression analyses to account for the hypothesized curvilinear relationship between achievement-related peer status and risk of eating pathology did not improve the fit of the model either (R-square change for all achievement-related peer-status variables 0.00; *p*(F change)> .05). In the model including the quadratic terms, affection-related peer status (social acceptance) at age 13 predicted eating pathology at 22 years after adjustment for other status domains (Table S6.3).

For the sake of parsimony, the quadratic terms were removed from the regression models. In the analyses without the quadratic terms, receiving more peer nominations for being good looking was associated with lower levels of eating pathology at age 22, also after adjusting for other status domains and for baseline eating pathology (Table 6.3). Higher peer popularity showed the same association, but this effect disappeared after other status domains were controlled for. Higher social acceptance emerged as a predictor for eating pathology at age 22 only in the model adjusted for other status domains. The use of the limited version of the T2 eating pathology score did not change any of the results, except that the association between peer status regarding physical attractiveness and later eating pathology was no longer significant in the model adjusted for other status domains (beta -0.09, *p*= .06).

Table 6.3 Self-esteem at 11 years and peer status at 13 years as potential risk factors for eating pathology at 22 years

	Beta (*p*)* Unadjusted for baseline eating pathology		**Beta (*p*)* Adjusted for baseline eating pathology**	
Self-esteem domains	**Unadjusted for other domains**	**Adjusted for other domains**	**Unadjusted for other domains**	**Adjusted for other domains**
Social acceptance	−0.07 (.05)	−0.02 (.54)	−0.07 (.05)	−0.02 (.54)
Physical appearance	**−0.17 (< .001)**	**−0.18 (< .001)**	**−0.16 (< .001)**	**−0.16 (.001)**
Academic performance	−0.02 (.50)	0.01 (.77)	−0.02 (.51)	0.01 (.75)
General	**−0.11 (.002)**	0.02 (.70)	**−0.11 (.001)**	0.01 (.86)
Peer-status domains				
Social acceptance	0.02 (.49)	**0.08 (.04)**	0.03 (.34)	**0.09 (.03)**
Physical attractiveness	**−0.09 (.01)**	**−0.10 (.04)**	**−0.08 (.03)**	**−0.10 (.046)**
Academic performance	0.03 (.44)	0.02 (.57)	0.02 (.50)	0.02 (.64)
Popularity	**−0.10 (.005)**	−0.07 (.10)	**−0.09 (.01)**	−0.06 (.14)

* All results are adjusted for gender; bold: $p < .05$

DISCUSSION

This study investigated whether self-perceived and peer-perceived social status among early adolescents – self-esteem at age 11 and peer status at age 13 - are associated with eating pathology in young adulthood. Data were drawn from a large cohort study which follows a community sample from early adolescence into adulthood. Both self-perception and peer-perception of physical attractiveness were inversely correlated with eating pathology at 22 years. Low peer popularity predicted eating pathology, but this effect disappeared when the other peer-status domains were controlled for. On the contrary, high social acceptance by peers emerged as a predictor of eating pathology only after adjustment for the other status domains.

To our knowledge, this is the first study that examined the role of peer status regarding physical attractiveness in the development of eating pathology. We found that receiving many nominations for good looks at age 13 was associated with low levels of eating pathology at age 22. Supporting evidence comes from a longitudinal study by Rosenblum and Lewis[23] showing that 13-year-old girls who were rated as attractive (as judged by study raters) were more satisfied with their bodies at age 18, regardless of body image at age 13 or physical attractiveness at age 18. The authors suggested that positive social feedback about physical appearance at age 13 lays the fundament of a positive body image that is relatively stable over time.[23] Negative body image is a very potent, well-supported risk factor for eating disorders.[3] It may not be surprising then that low self-esteem regarding physical appearance at age 11 poses a risk for the development of eating pathology.

A crucial developmental window around age 13 may explain why studies using older samples of adolescent girls[24] or female college students[25,26] found opposite results: higher objective ratings of physical attractiveness were correlated with higher levels of weight preoccupation cross-sectionally. Another explanation for the diverging results may be that physical attractiveness was judged by an average of seventeen classmates in our study, while this was done by only one research assistant in two of the other studies.[24,25] Furthermore, peer-group judgements of physical attractiveness reflect the daily social environment, which may be associated with other outcomes than fragmentary assessments by research assistants. Finally, in the studies by Davies et al.[25,26] care was taken to rate exclusively facial attractiveness, and not other factors such as weight, clothes or accessories; factors that may be of importance in peers' judgements about attractiveness.[27]

Although both self-perception and peer-perception of physical appearance predicted eating pathology, the two variables were virtually uncorrelated, indicating that these are largely different concepts. This is in agreement with the weak correlation (.24) between self-rated and judge-rated physical attractiveness found in a meta-analysis by Feingold.[13] He postulated that judge-rated physical attractiveness can be viewed as a status characteristic, while self-rated physical attractiveness is a facet of global self-esteem, and as such associated with positive mental health outcomes.[13] Based on our results, this might not hold true when the outcome is eating pathology. Though high global self-worth was inversely associated with later eating problems in the unadjusted model, this effect disappeared after the other self-esteem domains were controlled for. The effect of self-esteem regarding physical appearance did remain significant after controlling for the other domains. This indicates that, regarding risk for eating pathology, global self-worth depends on self-esteem regarding physical appearance rather than the other way round. In a more extreme form this constitutes one of the core features of the eating disorders anorexia and bulimia nervosa: an overvaluation of weight and body shape, and a sense of self-worth that is mainly - if not entirely – determined by weight and shape.[28] It must be noted though, that the SPPC measures a combination of appearance-related self-esteem (How happy are you with your looks?) and self-rated physical attractiveness (How attractive are you?), which are not identical concepts.[13]

Low peer popularity at age 13 predicted eating pathology at age 22, an effect that was accounted for by the positive correlation between popularity and peer status regarding physical attractiveness. Other studies found associations in the opposite direction: higher peer popularity was associated with more dieting behaviors cross-sectionally[10] and with more negative body-related cognitions one year later.[8] It is unlikely that the longer follow-up duration in our study explains the difference in results, because we also found an inverse cross-sectional correlation between peer popularity and eating pathology in girls. Wang and colleagues[10] reported a relationship between peer popularity and physical appearance: at about age 17, the bodies of popular boys and girls conformed to what is considered an 'ideal' body shape. The authors suggest that there may be a strong social reinforcement – being rewarded with popularity – for achieving a body shape that fits the current ideal.[10] Based on our results, however, good looks and concomitant popularity in early adolescence protect against eating pathology in young adulthood.

Contrary to our expectations, adolescents who were well-liked by their class mates at age 13 showed higher levels of eating pathology at age 22. Only one other

study[9] found that well-liked adolescent girls displayed more disordered eating behaviors and had lower body esteem than less-liked ones. The authors argued that girls who are well-liked might achieve high social acceptance because they rely heavily on the opinions of peers for their own self-worth and will go at length to be accepted. Girls whose self-worth is dependent on other people's judgements, either real or perceived, will actively conform to peer-group values and expectations – the thin-body ideal –, which may lead to body dissatisfaction and disordered eating behaviors.[9] Whether this process underlies the relationship between social acceptance and eating pathology in our study is hard to tell, and a question for future research. Another question is whether high social acceptance is a risk factor for eating pathology per se or reflects an underlying (personality) characteristic associated with increased risk, such as high interpersonal sensitivity,[29] socially prescribed perfectionism,[30] or externalized self-perceptions and self-worth.[9]

Strengths and limitations

To our knowledge, this is the first study on eating pathology examining the respective roles of self-esteem and peer status simultaneously. Strengths of this study include its longitudinal and community-based design, a sample including both male and female adolescents, and a long follow-up period, stretching eleven years from early adolescence to young adulthood. Response rates remained relatively high throughout the subsequent assessment waves. Data before the peak age of incidence of eating disorders were available[1], allowing us to make inferences on risk factors for eating pathology. Moreover, not only self-report data were used, but objective measures, such as length and weight, and data from other informants (parents and peers) as well, which is considered an important advance in eating disorder research.[31] Last, physical attractiveness was judged by a relatively large number of people, which probably increases reliability of the judgement.

There are several limitations to consider. The first concerns possible selection bias of the subsample used in this study: compared to the rest of the TRAILS cohort, the subsample had slightly better scores on almost all predictor variables, with the exception of appearance-related self-esteem, which showed no difference, and peer popularity, which was lower in the subsample. These differences may limit generalizability of the results to the general population. Furthermore, selective attrition of putative at-risk adolescents may have caused a restriction of range in specific self-esteem and peer-status variables, which in turn could have reduced the power to detect a significant correlation between those domains and later eating pathology. However, since differences between the subsample and the

rest of the TRAILS cohort are rather small, the consequences of selection bias are probably limited.

Second, since no baseline measure of eating pathology was available, we had to construct a proxy measure, with unknown sensitivity, specificity and validity. Furthermore, baseline eating pathology may be underestimated in part of the subsample, as not all indicator variables contributing to the sum score were complete for each participant. BMI had to be complete though in order to be included.

Third, the continuous symptom composite score of the EDDS was used, which has the advantage of increased power over categorical outcomes (e.g., eating disorder diagnoses). A drawback of this approach is that we cannot differentiate between eating disorder diagnoses, which may have different risk profiles. We believe that this might especially pertain to peer status. Though interpersonal difficulties are common in all eating disorders, specific patterns per eating disorder are discernable. For example, patients with restrictive eating pathology tend to avoid conflict, while patients with binge/purge pathology are more prone to conflict.[29] These specific characteristics of different types of eating pathology might influence peer status differentially.

CONCLUSION

In early adolescence, looking good - either in one's own eyes or in the eyes of peers - protects against eating pathology in young adulthood. However, being liked by peers might indicate a risk.

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SUPPLEMENTAL INFORMATION

Construction of proxy measure to control for baseline eating pathology

For both T1 and T2, the eating pathology score was based on three items regarding overeating, weight perception and possible compensatory behavior from the Youth Self Report (YSR items 53, 55 and 56g),[33] and one item on overeating from the parent-reported Child Behavior Check List (CBCL item 53),[33] which was included because studies have shown that parents add valuable information on this topic.[34] The T2 eating pathology score included four additional indicators based on items assessing body image and compensatory behaviors, which were not included in the assessment battery of T1. Adjusted BMI (%) was calculated by the formula: (actual BMI / median BMI for age and gender) x 100. The median age- and gender-related BMI scores pertained to Dutch children in 1997;[35] published data on median BMI assessed closer to the start of T1 (2001) were not available. An increase of one point in actual BMI corresponds with an increase of about 6% in adjusted BMI. Endorsement of an indicator added one point to the sum score. Some indicators were mutually exclusive (e.g., the presence of obesity or underweight). See Table S6.2 in Supplemental Tables for a more detailed description of the ten indicators for both T1 and T2.

SUPPLEMENTAL TABLES

Table S6.1 Comparison of final subsample and rest of total cohort*

Variables** T1	Mean (SD) Final subsample	Rest of total cohort	Difference *t*-test (*p*-value)
Age	11.09 (0.56)	11.12 (0.55)	$t = 1.10$ (.27)
Adjusted BMI	102.55 (14.94)	105.28 (18.60)	$t = 3.67$ (< .001)
Baseline eating pathology	0.11 (0.35)	0.18 (0.44)	$t = 3.93$ (< .001)
Self-esteem: social acceptance	3.10 (0.55)	3.04 (0.60)	$t = -2.61$ (.009)
Self-esteem: physical appearance	3.15 (0.60)	3.10 (0.68)	$t = -1.54$ (.13)
Self-esteem: academic performance	2.93 (0.49)	2.85 (0.55)	$t = -3.34$ (.001)
Self-esteem: general	3.37 (0.51)	3.31 (0.56)	$t = -2.45$ (.01)
T2			
Age	13.47 (0.51)	13.61 (0.53)	$t = 5.89$ (< .001)
Adjusted BMI	101.03 (15.16)	102.99 (17.99)	$t = 2.59$ (.01)
Baseline eating pathology	0.23 (0.53)	0.27 (0.58)	$t = 1.65$ (.10)
Peer status: social acceptance	0.56 (0.20)	0.54 (0.21)	$t = -1.22$ (.22)
Peer status: physical attractiveness	0.21 (0.21)	0.17 (0.18)	$t = -2.99$ (.003)
Peer status: academic performance	0.33 (0.26)	0.25 (0.23)	$t = -5.17$ (<.001)
Peer status: popularity	0.10 (0.12)	0.12 (0.14)	$t = 2.21$ (.03)
T5			
Age	22.22 (0.65)	22.30 (0.65)	$t = 2.58$ (.01)
Eating pathology	9.99 (9.93)	9.22 (10.14)	$t = -1.47$ (.14)

* Final subsample (n=699; 58.7% female) is part of the larger TRAILS cohort (n at baseline=2,230; 50.8% female), and selected on the basis of the availability of peer nominations by class mates at T2 in a subsample (n=1,007), and of complete data on adjusted body mass index (BMI) at the first (T1) and second (T2) assessment wave, completed measures of self-esteem at T1 and eating pathology at the fifth assessment wave (T5).

** Adjusted BMI (%) was calculated by the formula: (actual BMI / median BMI for age and gender) x 100. Baseline eating pathology: a proxy measure was created by means of indicators of eating pathology based on its core features: high or low BMI, binge eating, compensatory behaviors and distorted body image.[22] Endorsement of an indicator added one point to the sum score. The sum score could range between 0 and 4 for T1 and between 0 and 7 for T2. Self-esteem: Self Perception Profile for Children (SPPC)[17] domains social acceptance, physical appearance, academic competence and general self-worth. Scores could range between 1 (low self esteem) and 4 (high self-esteem). Eating pathology at T5: symptom composite score of the Dutch translation of the Eating Disorder Diagnostic Scale (EDDS)[19,21] by summing the raw items. Scores could range between 0 and 113 for women, and between 0 and 109 for men.

Table S6.2 Indicators of baseline eating pathology* at the first (T1) and second (T2) assessment wave**

Indicator	Variables***
T1 and T2	
Underweight	Adjusted BMI <85.0%
Obesity	Adjusted BMI >140.0%
Binge eating	I eat too much=very true or often true & Adjusted BMI >120.0%
Binge eating & compensatory behavior: vomiting	I eat too much=very true or often true & Vomiting= very true or often true
Distorted body image	I am overweight=very true or often true & Adjusted BMI <100.0%
Parent report: binge eating	Overeating & Adjusted BMI >120.0%
T2 only	
Binge eating + compensatory behavior: excessive exercise	I eat too much=very true or often true & Exercise to lose weight=yes
Binge eating + compensatory behavior: fasting	I eat too much=very true or often true & Breakfast frequency = never
Distorted body image	Body perception=much too fat & Adjusted BMI <100.0%
Distorted body image	Dieting=yes & Adjusted BMI <100.0%

* Baseline eating pathology: a proxy measure was created by means of indicators of eating pathology based on its core features: high or low BMI, binge eating, compensatory behaviors and distorted body image.[22] Endorsement of an indicator added one point to the sum score. The sum score could range between 0 and 4 for T1 and between 0 and 7 for T2.

** Of the TRAILS study.[15]

*** Explanation of variables:
Adjusted BMI: adjusted body mass index=(BMI / 50th percentile BMI for age and gender) x 100. Cut-off percentages for underweight (85%), overweight (120%) and obesity (140%) were based on Van Winckel & Van Mil.[32] Youth Self Report (YSR): items 53 (I eat too much), 55 (I am overweight) and 56g (Vomiting, throwing up without known medical cause).[33] Child Behavior Checklist (CBCL: item 53 (Overeating).[33] Exercise to lose weight, breakfast frequency, body perception, dieting: questions developed by TRAILS.

Table S6.3 Peer status at 13 years and risk of eating pathology at 22 years

	Beta (*p*) Unadjusted for baseline eating pathology		Beta (*p*) Adjusted for baseline eating pathology	
Peer-status domains	**Unadjusted for other domains**	**Adjusted for other domains**	**Unadjusted for other domains**	**Adjusted for other domains**
Social acceptance	0.02 (.49)	**0.09 (.03)**	0.03 (.34)	**0.10 (.02)**
Physical attractiveness*	-0.13 (.23)	-0.19 (.11)	-0.12 (.27)	-0.19 (.10)
Academic performance*	0.15 (.20)	0.16 (.16)	0.13 (.25)	0.15 (.19)
Popularity*	-0.04 (.64)	0.01 (.95)	-0.02 (.79)	0.02 (.84)

* Because achievement-related peer-status variables were hypothesized to have a curvilinear relationship with eating pathology at age 22, a quadratic term of each achievement-related peer-status variable (physical attractiveness, academic performance, popularity) was added to the regression model. All results are adjusted for gender; bold: $p < .05$.

Chapter 7

General discussion

In this thesis several aspects of the epidemiology of eating disorders in primary care and the community were investigated. This chapter provides a discussion of the main findings, methodological considerations regarding the empirical studies conducted, and implications for clinical practice and further research.

MAIN FINDINGS

A comprehensive review of the literature on the incidence, prevalence and mortality rates of eating disorders (Chapter 2) found that, while the overall incidence rate of anorexia nervosa (AN) remained stable over the past decades, there was an increase in the high-risk group of 15-19-year-old girls. It is unclear whether this reflects earlier detection of AN cases or an earlier age at onset. The occurrence of bulimia nervosa (BN) may have decreased since the early 1990s. All eating disorders have an elevated mortality risk; AN the most striking. Compared with the other eating disorders, binge-eating disorder (BED) is more common among males and older individuals.

The primary care study examining the incidence of AN and BN in the 1980s, 1990s and 2000s (Chapter 3) showed that the incidence of BN decreased significantly indeed over the past three decades, from 8.6 per 100,000 person-years in 1985-1989, to 6.1 in 1995-1999, and 3.2 in 2005-2009, while the incidence of AN remained fairly stable (7.4 per 100,000 person-years in 1985-1989, 7.8 in 1995-1999 and 6.0 in 2005-2009). Explanations were sought in sociocultural developments in the last 25 years to which BN is probably more sensitive than AN, such as a secular trend of increasing body mass index (BMI) of the general population, which may normalize overweight and thus decrease the pressure to counteract the effects of binge eating by compensatory behaviors; increased attention of the media and policy makers to eating disorders, lifting the taboo of – especially – BN, which is particularly surrounded by shame and secrecy; and a rise of prevention efforts and alternative sources of help, augmented by the rapid expansion of internet availability in the first decade of the new millennium.

In Chapter 4 the literature on the epidemiology, course and outcome of eating disorders in accordance with the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)[1] was discussed. The lifetime prevalence of DSM-5 AN among women is estimated to be up to 4%, and of BN 2%. In a cross-national survey, the average lifetime prevalence of BED was 2%. Several studies confirmed that the DSM-5 criteria for eating disorders effectively reduce

the proportion of the residual diagnosis 'eating disorder not otherwise specified' (EDNOS), although the magnitude of the reduction varies across the studies. Data on long-term outcome, including mortality, are limited for BED. Follow-up studies of BED are scarce; remission rates in randomized controlled trials ranged from 19 to 65% across studies. On a community level, 5-year estimated recovery rates for DSM-5 AN and BN are 69% and 55%, respectively. Little is known about course and outcome of BED in the community.

In the study examining the prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents (Chapter 5), the most common diagnoses among the girls were AN and BED, with a lifetime prevalence of 1.7% and 2.3%, respectively; and among the boys, BED (0.7%). In contrast, BN was relatively rare (lifetime prevalence among girls 0.8%). These results provide circumstantial evidence for the aforementioned hypothesis of declining BN rates due to a rise of isolated binge eating without compensatory behaviors. According to DSM-5 criteria, a minority of all diagnosed cases (15.5% of all eating disorder diagnoses; boys and girls combined) fell into one of the residual categories 'otherwise specified feeding and eating disorder' (OSFED) or 'unspecified feeding and eating disorder', compared with 64.4% for the DSM-IV residual category diagnosis EDNOS. Male and female adolescents combined, the lifetime prevalence of any eating disorder increased with 28.9% under DSM-5 criteria. This increase was mainly attributable to the inclusion of cases of BED that were subthreshold according to DSM-IV.

Next, the validity of the severity ratings for feeding and eating disorders was evaluated by examining their distribution in the community and their correlation with detection and treatment rates. As was to be expected for a community cohort, the severity of most cases was mild to moderate. A statistically significant association between severity and detection rate, and between severity and treatment rate was found. These results provide evidence for the validity of the DSM-5 severity ratings for feeding and eating disorders.

In the same community cohort of adolescents the role of self-perceived and peer-perceived social status in early adolescence as a potential risk factor for eating pathology in young adulthood was investigated (Chapter 6). Classroom social status in the domains of social acceptance, physical attractiveness, academic performance and popularity was assessed by means of peer nominations at age 13. Self-perception in corresponding domains was assessed at age 11. Both self-perceived and peer-perceived physical attractiveness were inversely correlated with eating pathology at age 22. Low peer popularity predicted eating pathology as well, but this effect was fully accounted for by the positive correlation between popularity

and peer status regarding physical attractiveness, and thus disappeared when the other peer-status domains were controlled for. On the contrary, high social acceptance by peers emerged as a predictor of eating pathology only after adjustment for the other status domains, showing that this effect was initially masked by social acceptance's positive correlation with peer status regarding physical attractiveness and popularity, which have an inverse relationship with eating pathology.

GENERAL DISCUSSION

The DSM-5 has two major ambitions: 1. to use a more dimensional approach in the diagnostic classification of mental disorders than DSM-IV, and 2. to organize the chapters of the DSM-5 according to developmental processes and life course, in order to increase clinical utility and to facilitate etiological scientific research by providing better and more flexible diagnostic concepts, which transcend the individual categorical diagnoses.[1] While a dimensional approach in diagnostic classification at first glance may look like a *contradictio in terminis* – how could one quantify something that is categorical? -, there are indeed several ways to describe a continuum of mental illness.[2] To begin with, there is the fundamental continuum ranging from mental health to mental illness. Within the domain of mental illness, degrees of severity can be defined, as reflected in DSM-5 by the introduction of a severity rating for disorders, including feeding and eating disorders, ranging from mild to extreme. In Chapter 5, we have shown that the severity ratings for feeding and eating disorders are valid in terms of their distribution in the community, and their correlation with detection and treatment rates. Age at onset did not differ between severity categories, reflecting that age at onset is probably more disorder- than severity-dependent. Neither did we find a correlation between severity and clinical recovery rate, which could be explained by the relatively young age of the sample, combined with a high threshold for clinical recovery.

A related example of dimensional thinking is the lowered diagnostic threshold for AN and BN in DSM-5, thus recognizing that subthreshold AN and BN according to DSM-IV – categorized as EDNOS - are not qualitatively different disorders from full-blown AN and BN, but just milder forms.[1,3] In the community cohort of adolescents (Chapter 5), applying the lowered diagnostic thresholds increased the female lifetime prevalence of AN and BN by 50% and 40%, respectively, compared with DSM-IV. In combination with the recognition of BED as a specific eating disorder in DSM-5, our results show that these alterations contribute to a substantial

reduction of eating disorder diagnoses in the residual category, which was an important goal for the Eating Disorders Workgroup for DSM-5.[4]

To regard mental disorders from a developmental perspective, where the same psychopathology can have multiple expressions during different phases of an individual's life, is yet another example of a dimensional approach.[2] This is best illustrated by a rather radical proposal by Fairburn & Cooper;[5] they suggest to eliminate the categorical diagnoses of (subthreshold) AN and BN altogether, and simply acknowledge that someone has a single eating disorder characterized by the transdiagnostic psychopathology of an overvaluation of weight and shape, which can have different behavioral expressions over time.[5] This proposal raises a fundamental question about diagnostic classification in psychiatry, namely, what is the purpose of such a system? Psychiatric diagnoses are phenomenological descriptions of symptoms, grouped into syndromes. Contrary to many somatic diseases, for mental disorders no additional 'objective' laboratory test (e.g., blood tests or neuroimaging) is available to confirm or rule out the diagnosis. While thus the underlying pathophysiology of most mental disorders eludes us still, the recognition of symptom patterns followed by diagnostic classification has proven to be effective in predicting course and prognosis of the disorder, and in deciding which interventions may be helpful.[1,6] Bearing in mind this fundamental purpose of informing treatment decisions, the question raised by Fairburn & Cooper[5] is: if the same psychological treatment works for patients from different diagnostic categories of eating disorders, what is the clinical utility of making the distinction anyway? That question lies beyond the scope of this discussion, but it illustrates how predictive validity is the central axis around which a diagnostic classification useful to clinicians should revolve.[6] Validity refers to the 'truth' of a diagnosis; in other words, if the diagnosis corresponds to a real, underlying clinical phenomenon, about which antecedent, concurrent and predictive characteristics can be described.[1] Reliability refers to the accuracy of a diagnosis; e.g., to what extent two clinicians examining the same patient arrive at the same diagnosis. General, unambiguous diagnostic criteria increase reliability but compromise the validity of a diagnosis, by summarizing and simplifying the complex and individual story of a patient's symptoms.[6,7]

What could be the consequences – either positive or negative - of a broadening of diagnostic concepts by lowering a diagnostic threshold? In other words, what are the costs and benefits of declaring more people ill? Clearly, the goal of lowering a diagnostic threshold is to identify more people who would benefit from any form of clinical intervention in order to prevent longstanding suffering or impaired role

functioning, somatic or psychiatric complications, or progression to a more severe course. An important question is where to put a diagnostic threshold in order to include – ideally - all people who would benefit from such interventions (high sensitivity), but avoid accidentally including people who would not (high specificity). In general, lowering a diagnostic threshold will increase sensitivity, but decrease specificity.[7] An example of a diagnostic threshold is the frequency and duration criterion of binge eating in BED: in DSM-5 binge eating needs to occur at least once a week for three months to warrant a diagnosis of BED; in DSM-IV a higher threshold was set: binge eating had to occur at least twice a week for six months. Furthermore, in DSM-5 a diagnosis of OSFED-subthreshold BED can be assigned to individuals with binge eating episodes who do not meet the diagnostic threshold of once a week for three months. Our results show that this lowered threshold for clinically significant binge eating is mainly responsible for the increase of almost 30% in the total number of eating disorder diagnoses.

The major downside of a mental disorder diagnosis is stigma and, especially when a diagnosis is assigned to mild deviations of normal behavior or to self-limiting responses to life's challenges, the risk of receiving unnecessary pharmacological treatment thwarting an individual's natural resources and resilience. On a public health level, a broadening of diagnostic concepts probably leads to an increase in expenses, since having an 'official' DSM diagnosis is often mandatory to get access to all sorts of insurance and government funds. In summary, by lowering a diagnostic threshold, certain unhealthy behaviors or transient responses to stressors run the risk of being unnecessarily medicalized, which may result in all sorts of unwanted costs for individuals, their environment and society.[*7]

Since the consequences of a mental disorder diagnosis are far-reaching, a crucial question is where to place the cut-off between, for example, unhealthy behavior and a mental disorder. Many psychiatric symptoms are aberrations of common behaviors and phenomena, either in intensity, duration, or both. This is most clear at the extreme ends of the continuum. Many people diet, but nobody will doubt that a 21-year-old woman with a BMI of 14 kg/m^2 who still feels fat and restricts her food intake, has AN and deserves treatment. Likewise is it not so difficult to dismiss a diagnosis of BED in a 30-year-old man who is usually so absorbed by his work that he forgets to have lunch and at the end of the day rapidly devours two meals and some candy bars, without displaying any of the psychological criteria of

* Something could be said as well about the consequences and potential risks of raising a diagnostic threshold, but since the criteria for all specific eating disorders in DSM-5 have been broadened, this discussion is limited to the consequences of lowering diagnostic thresholds.

BED, such as, for example, feeling disgusted with himself, depressed or very guilty after overeating.[1] The difficulties start in the grey area in the middle: where does 'normality' end and 'abnormality' begin? From a purely dimensional point of view, this question is irrelevant – if not impossible, because it assumes categories where none exist. From a clinical point of view, however, this question should be asked, in order to define groups of individuals who might benefit from clinical interventions, and exclude those who might not. On the continuum ranging from mental health to mental illness, cases of mild severity are closest to the ambiguous grey area in the middle. In the community cohort of adolescents (Chapter 5), mild cases constituted 53.2% of all feeding and eating disorder cases. Only a quarter of those were detected by the health care system, and a minority of 12.1% were treated. Would all mild cases benefit from treatment or any clinical intervention? Are mild cases on the slope to more severe pathology, or do they represent relatively common and self-limiting forms of eating pathology that do not require intervention? Our study could not answer this question, as clinical recovery rates did not differ between severity categories, probably accounted for by the relatively young age of the sample at diagnostic assessment. The DSM acknowledges the difficulty of making clinical decisions about the grey area where 'normal' and abnormal' meet, and tries to solve this by setting another threshold for a mental disorder: the presence of significant distress or disabilities in social or occupational roles.[1]

Normality in mental and physical functioning can be defined along population and individual norms.[6] Population norms are visualized in a bell-shaped curve showing that for any characteristic most people cluster around the mean.[6,7] Hence, normal is what most people are and - quite simplified - a deviation from the population norm and the individual norm can be regarded as a symptom.[6] Regarding the population norm only, the domains of symptoms are the extreme ends of the bell-shaped curve, which would theoretically result in an equal prevalence for all symptoms. The fact that prevalences of symptoms and mental disorders do differ despite the uniformity of the bell-shaped curve for population norms, could be explained by, for example, large variations in societal and individual tolerance for deviations from the norm, and by the syndromal constellation of symptoms, in combination with the additional criterion of significant distress and disabilities in role functioning, which constitutes the general definition of a mental disorder.[1] In the community cohort of adolescents (Chapter 5), confidence intervals of lifetime prevalence estimates of eating disorders to some extent overlapped, for example, of AN and BED among the girls, and of AN and BN among the boys, which renders the differences between those lifetime prevalences insignificant in this study. It is

likely, though, that a study with more power, that is: with a larger sample, would find significant differences between lifetime prevalences of eating disorders indeed.

For eating pathology and its concomitant features population norms have been shifting in the past decades, thus reshaping normality and abnormality. Take for example the rise of mean BMI of the global population over the past 40 years.[8] Does the fact that the majority of the US population is overweight or obese at present[9] mean that nowadays US citizens of normal weight have to be regarded as abnormal? Theoretically speaking, yes, but this example mostly serves to illustrate that normality does not equate with health, and that sometimes, the norm is a relative absence of health, or even illness. On the other hand, the normative increase in mean BMI, which to a lesser extent has affected the Netherlands as well, may actually be good news when it comes to risk of eating pathology: the norm of a fuller figure may be partly responsible for the decreased incidence of BN (Chapter 3); a hypothesis receiving support in Chapter 6, showing that adherence to group norms of physical attractiveness protects against the later development of eating pathology. This finding remained significant after adjusting for extremes of BMI (underweight and obesity). Whether group norms of physical attractiveness parallel a population norm (a fuller figure) or reflect an ideal norm (the thin-body ideal) is a question for further research.

The medicalization of normal variations and fluctuations in mental functioning is called diagnostic inflation, a process whose pitfalls are described by Allen Frances, chair of the DSM-IV taskforce and a prominent criticaster of the DSM-5, in his book 'Saving normal'.[7] Diagnostic inflation occurs when diagnostic labels are used – or misused – to serve interests that lie beyond the fundamental clinical purpose of a diagnosis, that is, of predicting course and outcome, and of informing treatment decisions. The main causes of diagnostic inflation are decreased societal tolerance for individual differences and eccentricity, and financial interests attached to mental disorder diagnoses, both for the individual (e.g., to get access to welfare and insurance funds) and for other parties (e.g., the pharmaceutical industry).[7] Furthermore, diagnostic inflation can occur when the Aesculapian authority of the skilled and experienced psychiatrist,[6] carefully evaluating a patient and assigning a diagnosis, is outsourced to less expensive - and less experienced – work forces. Examples include the use of lay interviewers in large-scale epidemiological studies, and general practitioners who are the first in line to evaluate and treat mental disorders before referral, if any, to a psychiatrist takes place. Another cause of diagnostic inflation is the creation of diagnostic hypes by experts with tunnel vi-

sion, the media producing sensational stories about mental disorders and, in the United States, direct consumer advertising by pharmaceutical companies.[7]

According to Frances, BED has the potential to become a future diagnostic hype. He questions the validity of BED by equating it with obesity and subsequently applies a social model of (mental) illness on BED/obesity: these conditions are the result of the food industry producing unhealthy yet abundantly and cheaply available foods, backed by governmental policy and subsidies.[7] Results from our review on the course and outcome of eating disorders (Chapter 4) and the study on the community cohort of adolescents (Chapter 5) contradict these statements about BED. First of all, BED and obesity are not identical: while 60.0% of adolescents with a lifetime diagnosis of BED were obese at age 19, only 16.5% of all obese adolescents (at age 19) had a lifetime diagnosis of BED. Furthermore, BED is distinct from obesity regarding concurrent validators (e.g., levels of psychopathology, weight and shape concerns and quality of life).[10] Also, the standard treatment for obesity - behavioral weight loss – does not work as well for binge eating as treatment with psychotherapy does,[11,12] providing evidence for the predictive validity of BED. Furthermore, BED may confer a risk of components of the metabolic syndrome,[13,14] over and above the risk attributable to obesity alone[13] (Chapter 4). Based on these results, Frances' approach of BED could be regarded as an example of diagnostic deflation, which could result in undertreatment, or offering an ineffective treatment, to a clinically significant problem. Finally, it could be disputed whether the application of a social model on BED/obesity is justified, or that it would be more appropriate to state that risk factors for overeating, and as such for obesity and BED, have increased.

Frances' criticism that BED is an example of a diagnostic hype in the making seems slightly unfair since BED was introduced in DSM-IV under his leadership. It nevertheless provides a good opportunity to examine whether the forces allegedly setting the stage for a diagnostic hype – experts, media and Big Pharma – will also play this role in the predicted dissemination of BED. Although it is impossible to forecast the future, the epidemiological history of the other specific eating disorders - AN and BN –, as examined in Chapter 3, may offer clues to BED's fate.

Up to now, the powerful searchlight of the pharmaceutical industry has not managed yet to illuminate an effective drug for the treatment of eating disorders.[15]

Hence, diagnostic hypes – if any – have not come from that direction**. The fact that eating disorders lack the 'patronage' of the pharmaceutical industry does not mean they dwell in some obscure corner of psychiatry though. On the contrary: perhaps more than any mental disorder eating disorders have been in the lime light – directed from many corners of society. In Chapter 3 we have discussed that from the 1990s onward, both AN and BN received a great – and increasing - amount of attention from the scientific field and the media. For example, the struggle of celebrities with eating disorders was discussed in the popular press, thus increasing awareness of these mental disorders among the public and potentially setting the stage for a diagnostic hype. This was augmented by reports of an 'epidemic' of eating disorders; a sensational terminology not limited to the popular press.[17] The primary care study examining the incidence of AN and BN in the 1980s, 1990s, and 2000s showed, however, that the incidence of AN remained remarkably stable in the past three decades, while the incidence of BN even sharply decreased (Chapter 3).

The ability to nuance sensational reports of an 'epidemic' constitutes one of the core values of well-conducted epidemiological studies with sound and rigorous methods. Such studies have proven to be invaluable in nuancing reports of epidemics of AN and BN, and they will hopefully serve this purpose for BED as well. There is, however, some aspect unique to BED which potentially makes this diagnosis not only more prone to diagnostic inflation, but to diagnostic deflation as well. BED may be a less accurate diagnosis than either AN and BN, as reflected in the key parameters of severity in DSM-5. BMI constitutes the key severity parameter for AN, which is highly objective in the sense that it can be measured by the clinician. For BN, frequency of purging episodes determines severity, which cannot be measured directly, but is an unambiguous pathological symptom that can be regarded as a categorical phenomenon: either you vomit, or you don't. Accuracy of diagnosis here depends on the willingness of the patient to disclose such – often shameful – behaviors to the clinician. For BED, severity is defined by the frequency of binge eating episodes. The core feature of binge eating is loss of control, a variable and subjective experience that cannot be assessed directly, but

** Eating disorder treatment centers offering residential treatment programs may fill the marketing gap left by the pharmaceutical industry. A recent article in the New York Times[16] discussed the rapid expansion of profit-based treatment centers for eating disorders in the United States since the introduction of the Affordable Care Act, enabling more patients with an eating disorder to afford treatment. Thus, this law increases the reservoir of potential 'consumers' of eating disorder treatments. The treatment centers directly advertise to consumers and clinicians with alluring descriptions and pictures of often spa-like facilities.

can only be addressed by circumstantial questions, partly reflected in the B-criteria of BED in DSM-5.[18] This inherent subjectivity of the core feature of BED increases the risk of an inaccurate diagnosis, and therefore of both diagnostic inflation and deflation.

Frances' criticism on BED also opens an interesting discussion about the role of societal forces and population norms in defining mental illness in general and eating disorders in particular. Regardless of whether his theory of a social model of BED/obesity is right, wrong or incomplete, it does give an example of which powerful societal and cultural forces may exert their influence in both creating the conditions and reshaping the norms of what is normal and what is abnormal; and consequently of what is (mental) illness. What cultural and societal forces may influence the occurrence of eating disorders? If we return to the notion that psychiatric symptoms are often aberrations of common behaviors, what pathological extensions of normality could eating disorders represent? These so to speak macro-level questions were studied on a micro level in Chapter 6, in which the role of self-perceived and peer-perceived social status in the risk of developing eating pathology was investigated. Like all social groups, adolescent peer groups have a hierarchical structure in which a member's status determines the amounts of respect, influence and attention assigned to that member.[19,20] Peer groups translate norms from a macro level (society) and apply them on a micro level. The other way round, peer group norms may offer insight into societal norms.

Physical attractiveness was the most salient domain of both self-perceived and peer-perceived social status in early adolescence regarding risk of eating pathology in young adulthood (Chapter 6). What does that say about the importance of physical attractiveness in the society at large? Present-day Western society is full of paradoxes: mean BMI has gone up, yet the beauty ideal of a thin body is a collective desire, its achievement demanding a restraint and self-control that is countered by the liberalism of doing whatever you like and your 'right' to indulge and enjoy yourself. We are pressed to distinguish ourselves and to be individualistic, yet the pressure to conform and to adhere to the prevailing norms is just as strong. Women have demanded and received equal opportunities to men regarding education and occupation, yet the societal demands on appearance and attractiveness are still most stringent for women, thus contradicting – if not refuting - that it is the inside that counts. These conflicting values and desires are capitalized on by all sorts of industries: the food industry coaxing us to eat more, the diet industry to eat less, the fashion and beauty industry promising us an improved look, and social media spreading a global and uniform sort of individualism.

Many of these paradoxes are played out in the field of physical appearance, not only because we are what we eat, and what we eat – or don't eat - affects our weight and consequently how we look, but also because these paradoxes are about how others perceive, judge and rank the part of us most readily visible to them - or how we think they do. It is therefore not surprising that physical attractiveness was the most salient domain of both self-perceived and peer-perceived social status regarding risk of eating pathology (Chapter 6). It is important to note that self-perceived and peer-perceived physical attractiveness are not necessarily two sides of the same coin. Looking good in one's own eyes may not mean a self-judged adherence to the prevailing cultural norms of physical attractiveness, but just as well being immune to them. Hence, it is truly the inside that counts, and peering through the looking glass is to see beyond reflections indeed.

METHODOLOGICAL CONSIDERATIONS

In this thesis, the occurrence of eating disorders was established at two levels of morbidity: primary care (Chapter 3) and the community (Chapter 5). Epidemiological studies at these levels of morbidity are important because the majority of eating disorder cases in the community do not seek help and are thus not detected by the mental health care system (Chapter 2). This underdetection may result in an underestimation and a distorted view (the so-called clinician's illusion) of the true scope and spectrum of eating disorders. Both the primary care and the community study employed a two-stage approach, which is considered the gold standard for relatively low-prevalent disorders such as eating disorders. In the first stage, a wide net of case identification was cast to avoid missing possible eating disorder cases (high sensitivity), followed by stringent case definition by eating disorder experts to establish or dismiss a final eating disorder diagnosis (high specificity). In the primary care study, the first stage - screening for new eating disorder cases - was performed by a nationwide network of general practitioners (GPs), serving a representative sample of the Dutch population. As such, we made use of the best qualities of GPs as central players in the health care and insurance system: they are the first to be consulted for any physical or mental problem, and they serve as gatekeepers to specialized care, thus being notified of all specialist consultations. Consequently, each GP has an overview of the diverse health problems of a large group of individuals in the community. The inherent generalist nature of their profession in combination with time constraints, however, make GPs less

equipped to establish accurate eating disorder diagnoses, so this part of the procedure was performed by the research team. In the community study, a cohort of 2,230 adolescents was screened for eating problems by means of data collected at a previous assessment wave. Again a wide net was cast to avoid missing possible cases: of the 312 adolescents considered to be at high risk for an eating disorder, a high proportion (95%) could be interviewed by experts, resulting in 58 adolescents with a definite lifetime eating disorder diagnosis.

Some fundamental methodological differences exist between the two studies, which are related to their respective purposes. In order to examine secular trends in the incidence of eating disorders, it is quintessential that the definition of disorder does not change over time, because a changed definition may confound any finding of a changed incidence over time. Therefore, the case identification criteria employed by the GPs in the primary care study were neither changed nor adapted since the start of the study in 1985, when only AN and BN were defined according to the then current DSM-III. A limitation of this methodological consistency is that new developments in eating disorder classification could not be incorporated in the study method. This mainly pertains to the DSM-IV residual category diagnosis EDNOS, which is the most common eating disorder in the community, a finding that was discussed in Chapter 2, and replicated in Chapter 5. The community study examining the prevalence of DSM-5 eating disorders, on the other hand, had the explicit purpose to investigate the epidemiological results of new developments in diagnostic classification, and hence used the most recent definition of eating disorders.

In the study examining the impact of self-perceived and peer-perceived social status in early adolescence on the development of eating pathology in young adulthood (Chapter 6), a continuous symptom score was used as the outcome measure (the overall symptom composite score of the Eating Disorder Diagnostic Scale).[21,22] This has the advantage of increased power over categorical outcomes (e.g., DSM-diagnoses), provided a linear relationship exists between the predictor variable and the outcome, which we assumed. A potential disadvantage is that we could not differentiate between eating disorder diagnoses, which may have different risk profiles. If we return to the notion of Fairburn & Cooper[5] of a transdiagnostic psychopathology of eating disorders – an overvaluation of weight and shape -, the use of a general eating pathology outcome measure seems valid. It must be noted, though, that Fairburn & Cooper[5] described predictive validity of the transdiagnostic approach, while antecedent validity was assumed in our study.

IMPLICATIONS

To prevent diagnostic inflation, Frances suggests a stepped-diagnosis approach, in which a definitive diagnosis is only assigned after the healing properties of time, social support and the placebo effect have not resulted in remission of the complaints.[7] The stepped-diagnosis approach may be a good choice when assessing mild forms of eating disorders, especially subthreshold forms of BED, which are classified in the DSM-5 residual category OSFED.

An important question arising from the general discussion, for which no clear answer yet exists, is how to handle mild cases of eating disorders in the community. Which of the mild cases will remit spontaneously, and which will progress to a more severe form? It would be worthwhile to investigate the predictive validity of severity ratings in terms of course and outcome, and to identify predictors of spontaneous remission. This especially pertains to BED, as not much is known about long-term course and outcome of BED at a community level. This knowledge will help to define individuals who may benefit from clinical interventions and prevent unnecessary treatment in those who will not. Severity ratings are designed for clinicians to track a patient's progress, but they serve as well in deciding how intense or invasive treatment should be. As such, severity ratings should go hand in hand with a stepped-care approach. Finally, the validity of the severity ratings for feeding and eating disorders could be further investigated by examining their correlation with other parameters, such as quality of life.

The finding that both self-perceived and peer-perceived physical attractiveness protect against later development of eating pathology provides evidence for the rationale of prevention efforts aiming to increase body satisfaction[23] and the use of peers in eating disorder prevention.[24] It would be of interest to investigate whether a discrepancy between self-perceived and peer-perceived judgement of physical attractiveness is associated with risk of eating pathology. Furthermore, whether peer group norms of physical attractiveness parallel a population norm (a fuller figure) or reflect an ideal norm (the thin-body ideal) would be another question for further research.

CONCLUSIONS

This thesis aimed to explore several aspects of the epidemiology of eating disorders in primary care and the community, with an emphasis on the effects of

changed diagnostic criteria, a changing sociocultural environment over time, and the impact of self-perceived and peer-perceived social status on the occurrence of eating disorders. It was found that the lifetime prevalence of any eating disorder increased with almost 30% under the DSM-5 criteria, and that this increase was mainly attributable to cases of BED that were subthreshold under DSM-IV criteria (Chapter 5). Since little is known about course and outcome of BED in the community (Chapter 4), it is as yet unclear what would be a cost-effective intervention strategy for mild or subthreshold cases of BED in the community. Furthermore, the DSM-5 criteria effectively reduced the proportion of the DSM-IV residual category diagnosis EDNOS, by making BED a specific eating disorder and by lowering the diagnostic thresholds for AN and BN (Chapter 5). The incidence of AN remained relatively stable over the past three decades, while the incidence of BN decreased substantially (Chapter 3). This finding illustrates the importance of sociocultural factors in the occurrence of eating disorders, especially for BN and perhaps for BED as well. Sociocultural factors and influences, such as the media, may also increase the risk of inaccurate diagnoses, diagnostic inflation and diagnostic hypes, to which BED may be more prone than the other specific eating disorders. On a micro level, that is: among peers, self-perceived and peer-perceived physical attractiveness protected against later development of eating pathology (Chapter 6). This finding was extrapolated to prevailing and often contradicting cultural values and norms in the society at large.

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Summary

Eating disorders are severe mental health problems with harmful consequences for physical and psychosocial health, which usually develop in adolescence. Their causes are relatively poorly understood. A classification of eating disorders can be found in the Diagnostic and Statistical Manual of Mental Disorders (DSM). The DSM is a reliable guideline to diagnose mental disorders. It offers a universal and transparent language to clinicians to inform treatment decisions in an individual patient; besides, it is well suited for examining the epidemiology and etiology of disorders. The fourth edition of the DSM, DSM-IV, specifies two eating disorders, anorexia nervosa and bulimia nervosa, and a residual category 'eating disorder not otherwise specified', which lists some examples such as binge-eating disorder. The majority of eating disorder cases in both clinical and community samples fall into the DSM-IV residual category, which is considered problematic. A major goal of the revision of the eating disorder section for DSM-5 has been to reduce the proportion of residual category diagnoses. For this purpose, the diagnostic thresholds for anorexia nervosa and bulimia nervosa were lowered, and binge-eating disorder was added as a specific eating disorder. Furthermore, DSM-5 includes a severity rating for disorders, ranging from mild to extreme, in order to help clinicians track a patient's progress.

Epidemiological studies provide information about the occurrence of disorders and may shed light on risk factors. Studies examining long-term trends in the occurrence of disorders may uncover risk factors that affect the society at large. In the case of eating disorders, this knowledge is salient since sociocultural factors are thought to play a major role. While examining long-term trends may provide knowledge about risk factors on a macro level (in the the society at large), studying a cohort of adolescents may offer insight into sociocultural risk factors on a micro level (e.g., at school or at home).

This thesis aims to explore several aspects of the epidemiology of eating disorders in primary care and the community, with an emphasis on the effects of changed diagnostic criteria, a changing sociocultural environment over time, and the impact of self-perceived and peer-perceived social status on the occurrence of eating disorders. A general introduction to eating disorders and epidemiology is offered in Chapter 1.

Chapter 2 provides a comprehensive review of the literature on the epidemiology of eating disorders, focusing on the basic epidemiological parameters incidence, prevalence and mortality rate. The incidence rate is the number of new cases of a disorder in the population over a specified period. The lifetime prevalence is the proportion of the population that has suffered from a disorder at any point in life.

From the 1930s until the 1970s, there was an increase of the registered incidence of anorexia nervosa in Europe. Since 1970, the incidence in Europe seems to have been rather stable. Less is known about long-term trends in the incidence of bulimia nervosa.

Chapter 3 examines changes in the incidence of anorexia nervosa and bulimia nervosa in the Netherlands during the 1980s, 1990s and 2000s, using data from a nationwide network of general practitioners, serving a representative sample of the total Dutch population. The incidence of bulimia nervosa decreased significantly over the past three decades, while the incidence of anorexia nervosa remained fairly stable. Explanations are sought in sociocultural developments in the last 25 years to which bulimia nervosa is probably more sensitive than anorexia nervosa.

Chapter 4 discusses the literature on the epidemiology, course, and outcome of eating disorders in accordance with the DSM-5. The first part describes the consequences of the revised diagnostic criteria for the incidence and prevalence of anorexia nervosa, bulimia nervosa, binge-eating disorder, and the residual diagnosis of not otherwise specified eating disorders. The second part reviews course and outcome studies regarding the three specific eating disorders in DSM-5 – anorexia nervosa, bulimia nervosa and binge-eating disorder. Several studies confirm that the DSM-5 criteria for eating disorders effectively reduce the proportion of the residual category 'eating disorder not otherwise specified'. In a number of studies, course and outcome of both anorexia nervosa and bulimia nervosa according to DSM-5 criteria did not differ significantly from course and outcome of these disorders as defined in DSM-IV. Data on long-term outcome, including mortality, are limited for binge-eating disorder. Moreover, the available data are solely based on clinical samples; therefore, little is known about course and outcome of binge-eating disorder in the community.

Chapter 5 reports on the prevalence and severity of DSM-5 eating disorders in a community cohort of Dutch adolescents. The most common diagnoses among the girls were anorexia nervosa and binge-eating disorder, and among the boys, binge-eating disorder. In contrast, bulimia nervosa was relatively rare. According to DSM-5 criteria, a minority of all diagnosed cases fell into one of the residual categories 'otherwise specified feeding and eating disorder' or 'unspecified feeding and eating disorder', compared with a majority of cases for the DSM-IV residual category 'eating disorder not otherwise specified'. Male and female adolescents combined, the lifetime prevalence of any eating disorder increased substantially under the DSM-5 criteria. This increase is mainly attributable to the inclusion of cases of binge-eating disorder that are subthreshold according to DSM-IV.

The validity of the severity ratings for feeding and eating disorders was evaluated by examining their distribution in the community and their correlation with detection and treatment rates. As was to be expected for a community cohort, the severity of most cases was mild to moderate. A statistically significant association was found between severity and the proportion of cases detected and treated by (mental) health care services. While only a minority of mild cases had been detected and treated, the opposite was true for cases of extreme severity. These results provide evidence for the validity of the DSM-5 severity ratings for feeding and eating disorders.

Chapter 6 investigates whether self-perceived and peer-perceived social status in early adolescence are associated with eating pathology in young adulthood. Data from the same community cohort of Dutch adolescents as described in Chapter 5 were used. Both self-perceived and peer-perceived physical attractiveness in early adolescence were inversely correlated with eating pathology in young adulthood. On the contrary, social acceptance by peers, that is: being liked, surprisingly emerged as a predictor of eating pathology.

The final chapter provides a discussion of the main findings, methodological considerations regarding the empirical studies conducted, and implications for clinical practice and further research. I conclude that the revised DSM-5 criteria effectively reduce the proportion of residual category diagnoses, whereas the total lifetime prevalence of eating disorders increases under the DSM-5 criteria. The severity ratings for feeding and eating disorders are valid in terms of both the distribution in the community and the correlation with detection and treatment rates by health care services. Furthermore, sociocultural values and norms, both in the society at large and in the proximate environment, influence the occurrence of eating disorders. This is illustrated by the finding that the incidence of bulimia nervosa decreased substantially over the past three decades, while the incidence of anorexia nervosa remained relatively stable, and by the finding that peer-judgements of physical attractiveness protect against future eating pathology.

Samenvatting

Eetstoornissen zijn ernstige psychiatrische aandoeningen met schadelijke gevolgen voor de lichamelijke en psychosociale gezondheid, die meestal ontstaan in de adolescentie. Over de oorzaken is nog relatief weinig bekend. Een classificatie van eetstoornissen is te vinden in de *Diagnostic and Statistical Manual of Mental Disorders* (DSM). De DSM is een betrouwbare handleiding om psychische stoornissen te diagnosticeren, die clinici een universele en transparante taal verschaft, waarna vervolgens beslissingen genomen kunnen worden over de meest geschikte behandeling van een individuele patiënt. Verder is de DSM zeer geschikt om de epidemiologie en etiologie van eetstoornissen te onderzoeken. De vierde editie van de DSM, de DSM-IV, specificeert twee eetstoornissen, anorexia nervosa en boulimia nervosa – en daarnaast een restcategorie 'eetstoornis niet anderszins omschreven', waaronder enkele voorbeelden worden opgesomd zoals de eetbuistoornis. De meerderheid van de patiënten met een eetstoornis, zowel in klinische als in bevolkingsstudies, valt in de DSM-IV-restcategorie, wat als een probleem wordt beschouwd. Een belangrijk doel van de revisie van de eetstoornissectie ten behoeve van de DSM-5 is geweest om het aantal restcategorie-diagnoses te reduceren. Hiertoe werden de diagnostische drempels voor anorexia nervosa en boulimia nervosa verlaagd en werd de eetbuistoornis toegevoegd als een specifieke diagnose. In de DSM-5 kan daarnaast de ernst van een stoornis worden gespecificeerd, waardoor clinici het beloop bij patiënten kunnen vastleggen. De ernstscores lopen uiteen van mild tot extreem.

Epidemiologische studies verschaffen informatie over de frequentie van stoornissen en kunnen inzicht geven in risicofactoren. Studies die langetermijn-trends in de frequentie van stoornissen onderzoeken, kunnen risicofactoren blootleggen die de gehele maatschappij aangaan. Voor eetstoornissen is dit soort kennis zeer relevant, aangezien socioculturele factoren waarschijnlijk een belangrijke rol spelen. Terwijl het onderzoeken van langetermijn-trends kennis kan verschaffen over risicofactoren op een macroniveau (in de maatschappij als geheel), kan het bestuderen van een cohort van adolescenten juist inzicht geven in socioculturele risicofactoren op een microniveau (bijvoorbeeld op school of thuis).

Dit proefschrift heeft als doel om verschillende aspecten van de epidemiologie van eetstoornissen in de bevolking en de eerste lijn te onderzoeken. Daarbij ligt de nadruk op de gevolgen van de veranderde diagnostische criteria, een veranderende socioculturele omgeving door de tijd en de invloed van sociale status op de frequentie van eetstoornissen. Een algemene inleiding in de eetstoornissen en de epidemiologie wordt verschaft in hoofdstuk 1.

Hoofdstuk 2 biedt een uitgebreid overzicht van de literatuur op het gebied van de epidemiologie van eetstoornissen. Het spitst zich toe op de fundamentele epidemiologische parameters incidentie, prevalentie en sterftecijfer. De incidentie is het aantal nieuwe ziektegevallen in de populatie over een bepaalde periode. De *lifetime* prevalentie geeft het percentage van de bevolking weer dat een bepaalde stoornis of ziekte nu heeft of ooit heeft gehad. Van de jaren '30 tot de jaren '70 van de 20e eeuw nam de geregistreerde incidentie van anorexia nervosa in Europa toe. Vanaf 1970 lijkt de incidentie in Europa redelijk stabiel te zijn gebleven. Er is weinig bekend over langetermijn-trends in de incidentie van boulimia nervosa.

Hoofdstuk 3 onderzoekt veranderingen in de incidentie van anorexia nervosa en boulimia nervosa in Nederland tijdens de jaren '80 en '90 van de vorige eeuw en in het eerste decennium van het nieuwe millennium. Hierbij werd gebruik gemaakt van data van een nationaal netwerk van huisartsen die met hun praktijk een representatieve steekproef vormen van de gehele Nederlandse bevolking. De incidentie van boulimia nervosa nam significant af in de laatste drie decennia, terwijl de incidentie van anorexia nervosa relatief stabiel is gebleven. Verklaringen worden gezocht in socioculturele ontwikkelingen in de afgelopen 25 jaar, waarop boulimia nervosa waarschijnlijk gevoeliger reageert dan anorexia nervosa.

Hoofdstuk 4 bespreekt de literatuur met betrekking tot de epidemiologie, het beloop en de uitkomst van eetstoornissen zoals geoperationaliseerd in de DSM-5. Het eerste deel beschrijft de consequenties van de gereviseerde diagnostische criteria voor de incidentie en prevalentie van anorexia nervosa, boulimia nervosa, de eetbuistoornis en de restcategorie van niet anderszins omschreven eetstoornissen. In het tweede deel worden studies besproken die het beloop en de uitkomst onderzochten van de drie specifieke eetstoornissen in de DSM-5, te weten anorexia nervosa, boulimia nervosa en de eetbuistoornis. Verschillende studies bevestigen dat de DSM-5-criteria voor eetstoornissen het aantal restcategorie-diagnoses 'eetstoornis niet anderszins omschreven' met succes hebben verkleind. In diverse studies waren het beloop en de uitkomst van zowel anorexia nervosa als boulimia nervosa volgens de DSM-5-criteria niet significant verschillend van het beloop en de uitkomst van deze stoornissen zoals gedefinieerd in de DSM-IV. Gegevens met betrekking tot uitkomsten op de lange termijn, inclusief sterftecijfers, zijn beperkt voor de eetbuistoornis. Bovendien zijn zij uitsluitend gebaseerd op klinische onderzoekspopulaties, waardoor weinig bekend is over het beloop en de uitkomst in de algemene bevolking.

In hoofdstuk 5 worden de prevalentie en de ernst van DSM-5-eetstoornissen in een bevolkingscohort van Nederlandse adolescenten gerapporteerd. De meest

voorkomende diagnoses bij meisjes waren anorexia nervosa en de eetbuistoornis en bij jongens alleen de eetbuistoornis. Boulimia nervosa was relatief zeldzaam. Volgens de DSM-5-criteria viel een minderheid van alle gestelde diagnoses in een van de restcategorieën 'andere gespecificeerde voedings- of eetstoornis' of 'ongespecificeerde voedings- of eetstoornis', vergeleken met een meerderheid van de diagnoses in de DSM-IV-restcategorie 'eetstoornis niet anderszins omschreven'. De totale *lifetime* prevalentie van eetstoornissen nam substantieel toe bij hantering van de DSM-5-criteria. Deze toename is vooral toe te schrijven aan enkele milde gevallen van de eetbuistoornis, die de diagnostische drempel van de DSM-IV niet halen.

De validiteit van de ernstscores voor voedings- en eetstoornissen werd geëvalueerd door hun verdeling in de algemene bevolking te onderzoeken en daarnaast ook hun correlatie met detectie- en behandelpercentages. Zoals te verwachten in een bevolkingscohort, was de ernst van de meeste ziektegevallen mild tot matig. Daarnaast werd een statistisch significant verband gevonden tussen de ernst en het percentage ziektegevallen dat was gedetecteerd en behandeld in de (geestelijke) gezondheidszorg. Terwijl slechts een minderheid van de milde ziektegevallen was gedetecteerd en behandeld, gold het omgekeerde voor ziektegevallen van extreme ernst.

In hoofdstuk 6 wordt onderzocht of sociale status in de vroege adolescentie, zowel zelf-ervaren als ervaren door klasgenoten, is geassocieerd met eetproblemen in de jongvolwassenheid. Hiervoor werd gebruik gemaakt van data van hetzelfde bevolkingscohort van Nederlandse adolescenten dat in hoofdstuk 5 werd beschreven. Er goed uitzien - zowel in de eigen ogen als in de ogen van klasgenoten – bleek te zijn geassocieerd met minder eetproblemen op jongvolwassen leeftijd. Opvallend is dat aardig gevonden worden door veel klasgenoten juist naar voren kwam als een voorspeller van eetproblemen.

Het laatste hoofdstuk biedt een bespreking van de belangrijkste bevindingen, de methodologische overwegingen bij de empirische studies en de implicaties voor de klinische praktijk en verder onderzoek. Daarin concludeer ik dat de gereviseerde DSM-5-criteria met succes de proportie eetstoornissen in de restcategorie reduceren, terwijl de totale *lifetime* prevalentie van eetstoornissen juist toeneemt bij het toepassen van de DSM-5-criteria. De ernstscores van voedings- en eetstoornissen zijn valide, gezien de verdeling in de algemene bevolking en de correlatie met detectie- en behandelpercentages in de gezondheidszorg. Verder beïnvloeden socioculturele normen en waarden, zowel in de maatschappij als in de directe omgeving, de frequentie van eetstoornissen. Dit wordt geïllustreerd door de bevinding

dat de incidentie van boulimia nervosa substantieel is gedaald in de afgelopen drie decennia, terwijl de incidentie van anorexia nervosa in diezelfde periode relatief stabiel is gebleven - en door de bevinding dat mooi gevonden worden door klasgenoten bescherming biedt tegen latere eetproblemen.

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"Kom! Lopende op blote voeten…"
(M. Vasalis)

Curriculum vitae

Frédérique Smink (Amersfoort, 1983) studeerde geneeskunde aan de Universiteit Leiden, waar zij in 2008 haar artsexamen behaalde. Vervolgens werkte zij zes maanden als zaal- en SEH-arts in het dr. L. Mungra Streekziekenhuis Nickerie te Suriname.

In 2009 startte zij met een gecombineerd traject van promotieonderzoek en de opleiding tot psychiater bij de Parnassia Groep in Den Haag. In het kader van haar promotieonderzoek hielp zij in 2009 mee met de dataverzameling van de TRAILS-studie in Groningen. Een deel van dit proefschrift is gebaseerd op data van TRAILS. In 2012-2013 nam zij zes maanden deel aan het Psychiatric Epidemiology Training Program aan de Mailman School of Public Health van Columbia University, New York. Daarnaast gaf zij in deze periode samen met een psycholoog wekelijks groepspsychotherapie aan eetstoornispatiënten in het Columbia Center for Eating Disorders, gevestigd in het N.Y. State Psychiatric Institute. De opleiding tot psychiater rondde zij af in 2013, waarna zij van oktober 2013 tot en met december 2015 als psychiater op de afdeling Angststoornissen van PsyQ Haaglanden heeft gewerkt.

Over haar ervaringen als arts in Suriname en als arts-assistent in opleiding heeft zij een aantal niet-wetenschappelijke artikelen en columns geschreven, welke verschenen zijn in respectievelijk Medisch Contact, het blad van de artsenfederatie KNMG, en De Psychiater, tijdschrift van de Nederlandse Vereniging voor Psychiatrie. Voor het artikel 'Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents', opgenomen in dit proefschrift, won zij in 2015 de prijs voor het beste artikel van het jaar van een jonge onderzoeker verschenen in het tijdschrift *The International Journal of Eating Disorders*.

Vanaf januari 2016 heeft zij zich geheel gericht op het afronden van dit proefschrift; na haar promotie zal zij het vak van psychiater hervatten.

Publications

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